

Dermatology
In General Practice

Contributors

LESTER W. BURKET, M.D., D.D.S.

Professor of Oral Medicine, Graduate School of Medicine,
University of Pennsylvania.

S. GORDON CASTIGLIANO, B.S., M.D., F.A.C.

Oncologist and Assistant Medical Director, Oncology Hospital, Associate in Oncology, Department of Radiology,
Graduate School of Medicine, University of Pennsylvania.

WILLIAM T. JOHNSON, M.D.

Associate Professor of Electrotherapeutics, Graduate
School of Medicine, University of Pennsylvania.

SAMUEL LISKER, M.D.

In charge of Peripheral Vascular Section, Department of
Medicine, Graduate Hospital, University of Pennsylvania.

JOHN B. LUDY, B.A., M.D.

(Deceased)

THEODORE MELNICK, M.D.

Assistant Pediatrician, Presbyterian Hospital, Visiting
Pediatrician, Philadelphia Hospital for Contagious Dis-
eases, Assistant Professor of Epidemic Contagious Dis-
eases, Graduate School of Medicine, University of Pen-
sylvania.

HERBERT W. WADE, M.D.

of the Leonard Wood Memorial Laboratory, C. Hooper
College, Philadelphia.

Dermatology

In General Practice

By

SIGMUND S. GRFENBAUM, B.S., M.D., F.A.C.D.

Professor of Clinical Dermatology and Syphilology University of Pennsylvania Graduate School of Medicine Dermatologist, Philadelphia General Hospital, Eaglesville Sanatorium, Philadelphia Psychiatric Hospital, Benninger Seashore Home Atlantic City; Betty-Barbarach Home Atlantic City; Bank Hospital, and Camden General Hospital, Connecticut Dermatologist, Mt. Sinai Hospital, Formerly Consultant Dermatologist to U. S. Veterans Administration Fellow American Academy of Dermatology and Syphilology Diplomat of American Board of Dermatology and Syphilology

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Dedicated to

JOHN B IUDY B A M D

*Without whose Contribution, and
That of His Friends, This Book
Would Not Have Been Completed*

Preface

The planning of this book as well as its organization was commenced by Dr John B Lusk in 1930. The stress of his extensive private and hospital practice and later of an illness which eventually proved fatal delayed its progress. However much of the manuscript containing contributions by Dr Castiglione and Dr Wade as well as numerous photographs had been collected by the spring of 1944. Sometime before this, I had been asked by my old friend Dr Lusk to carry the book through to completion. Despite almost thirty five years of special interest in diseases of the skin I approached the matter with trepidation especially because I was aware of how little we know about much of dermatology. The task, furthermore, was far more difficult than I had anticipated and was made even more so by a not inconsiderable physical handicap. In carrying through the mission however in writing and arranging the additional material including many illustrations, I have been constantly stimulated by my knowledge of Dr Lusk's kindnesses to many persons as well as to myself and because I felt that the material already gathered carried a message. The work of the contributors each expert in his special field aided materially in its fullness.

This book is designed for the busy practitioner the undergraduate and for all those who from time to time may require information on the major clinical diagnostic, and therapeutic facts about a particular dermatosis. The "Standard Nomenclature of Disease and Standard Nomenclature of Operations" has been followed almost entirely for the names of diseases.

Any classification of the dermatoses designed to make it easy for one to grasp the subject is difficult. Morphological and etiological classification and classification along anatomical lines have been the usual procedures. All are of value. All classifications, however reach a point where they break down, and the classification adopted here is no exception. As originally intended, the subject matter in this book has been arranged largely alphabetically—not altogether a new plan but one which it is believed will promote easy reference to its contents. Related dermatoses some on morphological and others on etiological grounds have been discussed together wherever possible. The dermatoses themselves have been treated as briefly as is consistent with the book's plan and design.

The context will be found to cover more than the mere essentials of diseases of the skin. Part of my function has been to provide a comprehensive survey of clinical dermatology and the material has been selected for its relationship to the needs of the practitioner. The various shifts in emphasis that the seasoned dermatologist will readily detect derive from this special approach. Similarly diagnosis and treatment are stressed throughout for these phases of dermatology are naturally uppermost in the minds of clinicians. Emphasis too has been placed upon the photographic presentation and it is believed that the reader will find this particularly valuable in a field that depends so heavily upon the visual aspects of morbid anatomy.

An outline of drugs and their application in skin diseases has been given. The formulas throughout have been successfully used in actual practice. The use of dithiol compounds, the newer agents in the therapy of ringworm and the use of the sulfonamides and of the antibiotics, especially penicillin, have been discussed in detail. The treatment of urticaria and allied conditions with the new antihistaminics has been fully described. Psychosomatic disturbances as they express themselves in the skin have received more attention than usual.

No attempt has been made to give credit to all those clinical and laboratory research students who have contributed to the advancement of our knowledge of diseases of the skin. The help and inspiration received from the published works of others are acknowledged with appreciation and thanks. Free use of numerous publications on diseases of the skin and syphilis has been made. Notes from lectures delivered while I was at L'Hopital St. Louis Paris in 1919 to 1920 given under such dermatologic masters as Louis Brocq, Joseph Darier, of Hudebo, Jeanselme, Milian, Gougerot and Ravaut have been freely consulted. I owe much to them as well as to the excellent collection of moulages found in the Museum of L'Hopital St. Louis. It is hoped that acknowledgment made at this point will be acceptable although I feel it is far from adequate recognition of those who spent many long hours in the production of original work.

The material on cancer of the skin was contributed by Dr. S. Cordón Castiglano. The article on leprosy was written by Dr. H. H. Wade. The fine standard of preventive medicine enforced upon our troops overseas appears to have been a fruitful one. Although leprosy has a long incubation period it does not appear now as if it and such tropical diseases as yaws

and leishmaniasis will constitute a significant hazard in the United States such as had been feared when this book was planned. This likewise holds true for the various dermatoses which appeared so frequently in the tropical environment in which our men found themselves. The article on leprosy, extensive when originally contributed, has been allowed to remain. Since some of our men have served in areas where leprosy is endemic, it is safe to predict that a certain number of them will become victims. Dr. Samuel Lasker contributed the articles on Raynaud's and Buerger's diseases and on varicose veins and their complications. Dr. William T. Johnson contributed the article on diathermy. Dr. Theodore Melnick contributed the material on the exanthemata. Dr. Lester W. Burket contributed the article on diseases of the oral mucous membranes.

It is hoped that this book will be of real value to those for whom it is intended and that they will find its contents helpful in their cutaneous diagnostic and therapeutic problems. The author wishes to take this opportunity to thank Dr. Jacques P. Guequierre and others for the use of illustrations, and to express special appreciation to Dr. Carroll S. Wright not only for the use of his illustrations and for his many helpful suggestions, but for his kindness in carefully reading both galley and page proofs. Finally, I should like to thank Mr. Wendell H. Grenman and Mrs. Margery Van Taube of the medical editorial staff of the F. A. Davis Company for their helpfulness and active cooperation in the birth of this book. They contributed much to my pleasure in carrying the work through to completion.

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Dermatology
In General Practice

ANATOMY AND PHYSIOLOGY

The skin (integumentum) is an elastic fibrous tissue covering the entire body. It serves as an agent for protection, temperature regulation, excretion, and reception of stimuli. The nerve, blood, and lymph supplies are located in the connective tissue. Appendages of the skin are hair, nails, and glands.

The skin is being constantly regenerated by growth from the strata underlying the skin surface and its several layers differ in histologic architecture and chemical composition.

The skin consists of two main layers; namely the epidermis (the superficial epithelial covering) and the derma or corium (the true skin).

Epidermis This is a development from the embryonic ectoderm. It is originally one layer of cells, but at the end of the first embryonic month it consists of two layers; the periderm or outer layer and the inner layer. The outer layer disappears and from the inner layer develop all layers of the adult epidermis.

The epidermis consists of cornified stratified squamous epithelium and is divided into five avascular distinct layers which are named from within outwards: the *stratum germinativum*, the *stratum malpighii*, the *stratum granulosum*, the *stratum lucidum*, and the *stratum corneum*.

Stratum Germinativum (Basal Cell and Innermost Layer) This consists of a layer of small basal columnar cells situated parallel to one another effecting a palisade appearance. This basal cell layer rests upon the basement membrane which lies directly over the corium and gives off serrated processes which indent the corium. These cells show signs of

mitosis which is followed by cell division. The basal cells contain granules of pigment (melanin) which produce the color of the skin. In this layer there are granules of the albuminoid keratohyalin as well as the chemical substance glutathione which gives the sulfhydryl (SH) group reaction.

Stratum Malpighii (Stratum Spinosum, Rete Mucosum, Prickle Cell Layer) This rests on the basal cell layer of the stratum germinativum. This layer consists of polyhedral flattened cells connected by spinous processes forming connecting bridges in the intercellular spaces, hence the appellation prickle-cells.

Ranvier compared this prickle cell layer to one vast cell with numerous nuclei. The intercellular spaces contain fluid which bathes the spinous processes and aids in cell metabolism.

Stratum Granulosum (Granular Layer) This layer consists of flattened coarsely granular cells. They are a transition from the prickle-cells of the stratum malpighii. The upper row of the prickle-cells are flattened and their nucleus is smaller. These cells contain an abundance of keratohyalin and eleidin.

Stratum Lucidum This stratum lies above the stratum granulosum and is best seen in sections from the palms and soles where the horny layer is overdeveloped. It appears as a transparent, clear more or less structureless layer containing several layers of flattened angular cells with refractile cytoplasm and indistinct nuclei. The cytoplasm cell appears swollen with fat (eleidin) droplets and occupies the whole cell. The granules of keratohyalin fuse in this layer.

Stratum Corneum (Horny Layer)

This lies externally and contains a compact mass of variable thickness of fused nonnucleated flat horny cells presenting vacuoles. The lamellae near the surface are readily separated from one another.

The thickness of the stratum corneum depends upon the amount of protection required in the different regions of the body—being thinnest on the face and on the flexor surfaces of the extremities and thickest on the palms and soles.

The stratum corneum may be conveniently divided into three zones: the lower zone presents closely packed cells containing a large amount of a waxlike substance, making it waterproof. This is covered by a looser zone consisting chiefly of a cell membrane in the state of advanced keratinization. The outer zone is more dense because the keratinized cells are closely packed and are constantly desquamating.

The epidermis unites with the underlying corium by prolongations or rete cones which dip down into the corium.

Corium (Dermis, Cutis, or Cutis Vera) This intervenes between the epidermis and the subcutaneous tissue with which it lies in intimate relationship.

It is derived from the embryonic mesenchyme and develops into a fibroelastic covering of connective tissue.

The corium is arranged in three distinguishable layers: the uppermost layer (the papillary body) lies immediately below the epidermis. It derives its nutrition from the papillary body. The papillary body consists of fine connective tissue fibers and loosely arranged cells which fit into the rete cones (downward serrations of the epidermis). The portion lying between the rete cones is known as the papillae and contain the nerve terminals, capillary loops, and the wide lymph spaces. It is in this papil-

lary layer and in the lymph spaces that the majority of the deeper pathological conditions of the skin take place.

The reticular layer of the corium consists of large coarse variously arranged collagenous bundles of connective tissue and comprises the bulk of the corium. The upper layer below the papillary body is made up of closely arranged fibers running in horizontal bundles. In the lower layer the fibers are more coarse and include the sweat glands and the hair roots.

Hypodermis This is the subcutaneous tissue immediately underlying the reticular layer of the corium. It consists of a network of interlacing and anastomosing fasciculi and bundles of connective tissue less densely arranged than those in the corium and encloses irregular spaces containing quantities of fat cells. It is essentially a continuation of the reticular layer and is attached to the fascia of the underlying muscles and deeper structures.

Skin Appendages These are modified epidermal tissue developing in early fetal life from solid invaginations of the epidermis descending into the corium where they differentiate into the respective skin appendages identified as hair, nails, sebaceous glands, and sweat glands.

Hair Hair is a horny structure that varies in cross section from circular to angular. The hair develops in the hair follicles as an invagination of the epidermis within the corium which sends out an outgrowth consisting of a small capillary loop to form the hair papilla which embraces, in turn, the hair bulb. The cells of the hair papilla proliferate and differentiate into horny tissue recognized as hair which grows upward in the follicles.

The adult hair is partly composed of keratin and melanin and consists of a

shaft and a bulb. The shaft consists of the cuticle, a cortex, and medulla. The cuticle is the external covering of the hair-shaft and consists of a mass of horny plates lying along the shaft.

The cortex contains flattened, elongated, cornified, and pigmented epithelial cells which are separated by a variable number of air spaces.

Microscopically the medulla appears as an irregular central stripe depending upon the number of air spaces present.

Human hair consists of 18 to 21 per cent of cystine which is contained in the keratin. Therefore, the addition of cystine to a diet deficient in this amino acid favors the growth of hair.

Sebaceous Glands. These are single or branched saccular tubules which are lined with stratified squamous epithelium. These glands lie in the lower portion of the corium and occur everywhere in the body except the palms and soles. They exist in two forms: grouped about the hair follicle into which they empty by a common duct, and a free form occurring in the hairless portion of the skin (e.g., glans penis, labia minora, and dorsum of distal phalanges). They arise from epidermal invaginations in connection with the developing hair by undergoing a peculiar fatty metamorphosis which terminates in the formation of sebaceous glands excreting sebum. These glands are well supplied with lymphatics, capillaries, and nerves derived from the subpapillary network and the sebum is discharged by contraction of the arrectores pilorum muscles.

Sweat Glands. Sudoriferous or coil glands develop in the same way as the sebaceous glands except that they are narrower and descend deeper into the lower region of the corium where they lengthen to maximum development by coiling. The sweat gland forms a con-

vulated globular mass which terminates in a blind tubule, the uppermost end of the tubule serving as the duct. The duct is lined by one or two layers of cells. The coiled part of the gland is the secreting portion and is lined with a single layer of cuboidal cells. The sweat gland opens with an orifice on the cutaneous surface or into the neck of the hair follicle.

The duct pursues a fairly straight course through the corium and between the papillae into the rete where it tends to form a tortuous channel. This channel occurs between the epidermal cells above the stratum germinativum so that the sweat may freely communicate with the interepithelial lymph spaces. The duct finally reappears on the cutaneous surface with a funnel-shaped opening called the "sweat pore."

The sweat glands are not only concerned with the excretion of sweat but they excrete also a certain amount of fat which according to Unna, plays an important part in lubricating the skin. The tubular coil of the sweat gland is invariably placed in immediate relation to a fat lobule from which it presumably derives the fatty substance necessary for lubrication. This is evident on the palms and soles where no other glands but coiled glands are present.

Nails. These are elastic platelike appendages measuring about 0.5 mm in thickness, originating as an invagination from the epidermis and covering the dorsal aspect of the terminal phalanges of the fingers and toes. They are formed to protect the extremities of the fingers and toes, to safeguard the sense of touch, and to play the important part of prehension of small objects.

Chemically the most important constituent of the nail is keratin. Keratin is an albuminoid containing 3 to 5 per

cent of cystine (sulfur) Lapidis occur in the nails in the form of cholesterol and help to maintain the elasticity of the nails The remaining chemical elements are made up of water calcium phosphorus, carbonates, and small amounts of arsenic

The skin of the dorsal aspect of the terminal phalanx is covered by the nail plate, the substance of which corresponds with the horny layer of the epidermis and is composed of layers of flattened horny cells The nail plate rests laterally in the nail grooves which are formed by the fold of skin The outer surface of the nail is shiny translucent smooth and pinkish in color while the under surface presents numerous parallel ridges running longitudinally which fit into similar depressions of the contiguous nail bed The nail bed acts as a nutritive agent to the nail plate and holds it firmly in place.

The lower part of the nail invagination comprises the nail matrix and extends from one lateral border of the nail body to the other It is composed of a basal layer of cuboidal cells and several layers of polygonal cells filled with fine granules of keratohyalin which give it the peculiar white cloudy color The anterior part of the matrix which appears as a half moon formation is called the "lunula" or "lunule."

Capillaries are particularly abundant on the nail bed and under the nail root The nail root is the portion of the nail plate which is embedded in the corium and which supplies the nail matrix with nutrition.

The matrix is the only reproductive part of the nail

Under normal conditions, the nail plate grows continually from the matrix to the free edge in from 120 to 180 days. The average daily growth of the nail is

0.1 mm. per day though the growth is more rapid in summer than in the winter season

Blood Vessels Those of the skin form a highly vascularized network lying only in the corium and subcutaneous tissue The epidermis has no vascular supply The blood vessels are arranged in two horizontal planes, namely the deep and superficial plexus The deep plexus is a coarse network and lies below the corium supplying the sweat glands and the papillae of the hair roots. The superficial plexus, a delicate network of vessels, lies above the corium and supplies the papillary body and gives off branches to individual papillae. Each papilla is therefore supplied with a fine capillary loop

Nerves of Skin These nerves are of cerebrospinal and sympathetic origin, serving as sensory vasomotor and motor elements containing medullated and non-medullated fibers There are two plexus of nerves; one lies in the subcutaneous tissue and sends branches to the upper portion of the corium Single fibers terminate in a special nerve-end body which contains a convoluted network of nerve fibrils These nerve-end organs are represented by (1) Pacinian corpuscles which are the larger of the nerve-end organs. They are ovoid in shape and are found in the lower strata of the corium. (2) Meissner's corpuscles, or touch corpuscles are round or ovoid and are found in the papillae These corpuscles are most plentiful where the hairs are absent on the terminal phalanx of the fingers (3) Krause's corpuscles consist of thin club-shaped elements and are found on the clitoris, penis, the soles, and the edge of the eyelids. (4) Ruffini bodies, or spindles, are long oval-shaped elements and are found in a lower stratum of the corium on the palms and

Anatomy and Physiology

soles and are thought to be concerned with heat perception.

The skin is abundantly supplied with motor and vasomotor nerves, the former being concerned with all glands that contain a muscular coat and may be found in relation to the hair follicles and in numbers around the sweat glands.

Muscles of Skin These are of the involuntary or smooth, variety the fibers of which make up bundles that terminate high up in the corium forming the *musculi erectori pilorum*. These muscles are attached to the hair follicles by bands of elastic tissue which, on contracting, erect the hair and express sebum from the sebaceous glands. Smooth muscle fibers are also found in the walls of the blood vessels and about the sweat glands.

Striated muscles of the skin are few in number and are found in subcutaneous tissue and the corium of the face and neck but play no part in the pathology of the skin.

Skin Pigment This depends upon the vascular content of the skin, the thickness of the stratum corneum, and a complex chemical substance known as melanin. Melanin is produced by the basal cell layer of the stratum germinativum by action of oxidising enzymes in the epidermal melanoblasts, a colorless substance allied chemically to tyrosin and epinephrin. The exact nature of melanin remains obscure but the substance acts as a protection against the harmful effects of heat and light. Melanin is found in the form of orange-yellow to black granules; they collect about the cell nucleus at the cell periphery.

Pigment cells have been demonstrated by the method of Bloch (Dopa reaction) to be contained also in wandering dendritic cells which occur throughout the

basal cell layer of the epidermis and in wandering phagocytes of the corium.

In addition to melanin, the presence of a lipochrome has been described in the skin and hair to which is attributed the characteristic red coloration which is often seen in hair.

Pigment formation can be augmented and increased by exposure to sunlight, roentgen rays, chronic inflammation, etc.

Functions of Skin The skin serves as an agent for body protection, it helps regulate body temperature, plays a rôle in eliminating body waste and serves as a receptor of external stimuli.

As a protective agent, the horny cells of the epidermis contain a waxlike substance which "waterproofs" the cutaneous surface of the body. It prevents extensive transudation of fluid through the skin and materially aids as a defense against micro-organisms. Micro-organisms proliferate freely and play their pathogenic rôle when the skin is deprived of its natural oils.

The sebaceous secretion makes the surface of the body oily thus preventing it from becoming sodden.

The skin acts as regulator of temperature by virtue of the dilatation and contraction of the cutaneous vessels and action of the sweat glands. It is only when the surrounding temperature reaches the height of 90° to 95° F that the sweat glands begin to operate vigorously and sufficiently to make beads of sweat appear on the skin surface. The normal amount of sweat secreted per day is said to be from 500 to 2000 cc., evaporation keeping pace with the secretion.

The skin plays its rôle in eliminating body waste and in cleansing the skin, excreting about 9 gm. of carbon dioxide a day (800 gm. by lung) and small

amounts of other wastes, such as various salts and urea

As a receptor of stimuli the skin maintains four sensations, namely *heat cold touch* and *pain* which are distributed

in an irregular manner over the skin. The application of cocaine to a cutaneous nerve causes the disappearance of the sensations in the following order: *pain cold heat*, and *touch*.

PRINCIPLES OF THERAPY IN DERMATOLOGY

In the therapy of skin diseases, it is well to remember that sometimes cure is obtained only by destroying or removing the cause; in others, by treating only the inflammatory phenomena resulting from the action of the irritant. Thus, in a tertiary syphilitic destruction of the *Treponemata* or raising the local resistance by specific or nonspecific agents is followed by healing. In dermatitis venerea removal of the irritant or of the skin from contact with it must always be accompanied by treating the existing inflammation. The epidermis, with the aid of this treatment, tends to push off the irritant. Sometimes the cause can be treated only after the result of its action has been controlled. Thus, in inflammatory or secondarily infected ringworm attempts to destroy that organism or the secondary invaders usually lead to over treatment. In other instances, the very action of the irritant (kernon, chemicals) produces a sufficient reaction to bring about a cure. In these cases, the therapeutic aim is to aid nature to complete its own defense.

Therapy in dermatology is highly specialized and its success depends on (1) a correct diagnosis; (2) the choice of medication, and (3) the mode of application. The causative factor in the majority of skin diseases is in or on the skin itself; therefore, external therapy is the rational method of treatment. Acute skin conditions are benefited by soothing applications while chronic skin lesions are benefited by stimulating agents. The following rules should be observed:

1 Crusts and scales covering the primary skin lesions must first be removed

A prolonged warm bath to which several ounces of carbonate of potash has been added is often all that is necessary. On the scalp, paraffin oil is usually sufficient to remove accumulated scales and secretion. Equal parts of petrolatum and lead plaster spread thickly on cloth is very efficacious and at the same time has an antiphlogistic action on inflamed areas. Some authors consider the borie-starch poultice the most satisfactory application; it is soothing as well as effective in removing any products of disease. It is made as follows: One teaspoonful of borie acid powder is mixed with four tablespoonfuls of either wheat or corn starch, and enough cold water is added to give the mixture the consistency of cream, a pint of boiling water is added and the mixture is stirred until a translucent jelly results. When the mixture is cold, spread on muslin in a layer about $\frac{1}{2}$ inch thick. This should be renewed four times each day.

2 Ointments should never be used for treating weeping skin lesions. Weeping surfaces are treated by applying such lotions as a 1 per cent aqueous solution of aluminum acetate, ichthammol or a 0.25 per cent aqueous solution of silver nitrate or by a bland powder.

3 Ointments are also contraindicated on an acutely inflamed skin where the temperature of the involved skin is higher than that of the adjacent skin. Evaporating lotions or cold starch poultices are the best therapy for the treatment of an acutely inflamed skin. Ointments and oils leave a film over the inflamed skin and interfere with the normal regulation of body heat.

amounts of other wastes such as various salts and urea

As a receptor of stimuli, the skin maintains four sensations, namely *heat cold touch* and *pain* which are distributed

in an irregular manner over the skin. The application of cocaine to a cutaneous nerve causes the disappearance of the sensations in the following order: *pain cold heat* and *touch*.

lowed. Overfeeding is the chief danger in diseases of the skin. Frequently underfeeding will produce a beneficial result, especially in cases of chronic eczema. It is better to eat more frequently than to partake of heavy meals. An occasional "fast day" during which buttermilk is the sole article of food, is helpful in cases of chronic skin disease.

The question of washing the skin is important. Soaps owe their detergent (cleansing) action to their alkalinity which makes them harmful for certain normal and diseased skins and helpful for others. Some skin diseases, like psoriasis, derive benefit from frequent bathing in warm water and soap. Washing in water alone is not harmful. It is the soap and friction with which it is applied which act as irritants. Acute and weeping eczema is benefited by washing in warm normal salt solution.

Internal Medication. It would not serve any useful purpose to devote a section to the pharmacological action of the many internal remedies useful in the different skin diseases. Some of those in common use which are given orally include:

Arsenic. This is given either as Fowler's solution (potassium arsenite solution) or as arsenic trioxide. It is an alterative, hematinic, and tonic. It is probably the most widely used and abused remedy in dermatology. It is of definite value in the treatment of chronic psoriasis, dermatitis herpetiformis, pompholyx, pemphigus vulgaris, lichen planus, and mycosis fungoides. An excellent mode of administration is as follows:

Fowler's solution (100 potassium arsenite) 30.0
 Tr. psyllium comp. 30.0
 See For 5 days, take 5 drops in little
 after breakfast. For the succeeding 5
 days, take the same dose, take 10 drops. Increase
 the dose 5 drops at the end of each five-day
 period, until maximum of 30 drops is reached.

Then decrease the doses every five days until the 5-drop dose is again reached, extending the treatment over a total of seventy-five days.

The patient should report once weekly for detection of signs of intolerance. If they develop, the dose for that patient is at maximum and should gradually be reduced. At least 10 months should elapse before second cycle of treatment.

Calcium. This is given orally as calcium lactate, calcium gluconate, calcium chlorate, and calcium sulfide. It is useful as a prophylaxis in serum sickness and in the treatment of chilblains, urticaria, and verrucae plana. Calcium sulfide is especially indicated in the treatment of boils, abscesses, and pustular acne. Calcium peroxide is used externally in the treatment of infected wounds, while calcium permanganate is useful in the treatment of rodent ulcers and inoperable carcinoma.

Salts of Mercury. These are given orally in such diseases as syphilis and lichen planus. The cyanide of mercury is less irritating than mercuric chloride and is given orally in doses of 4 to 8 mg ($\frac{1}{16}$ to $\frac{1}{8}$ grain).

Dilute Hydrochloric Acid. This is given in the treatment of acne rosacea, pellagra, and allergic skin diseases.

Iodine. In the form of potassium iodide, this is useful in the treatment of tertiary syphilis, actinomycosis, blastomycosis, and sporotrichosis.

Sulfonamides. These include, among others, sulfanilamide, sulfapyridine, sulfathiazole, and sulfadiazine. The least toxic as well as the most active of these is sulfadiazine. It is useful in all pyrogenic dermatoses as well as some of the virus diseases, especially lymphogranuloma venereum. Its use is indicated in the treatment of erysipelas, erysipeloid, cellulitis, impetigo, furunculosis, and secondary pyrogenic infection. Allergic response, toxic manifestations and fatality

4 Strong antiseptic medication is avoided in treating suppurating lesions of the skin. It is better practice to begin treatment of suppurating skin lesions by applying a hot saturated solution of boric acid or by a calamine lotion to which 1 per cent ichthyol is added.

5 Adhesive plaster is never applied in the vicinity of eczematoid and pustular eruptions.

6 Gauze is irritating to an inflamed skin and should not be used unless kept constantly moist. Bleached muslin and rayon are better.

7 Tar ointment and lotions containing inert powders should not be applied on hairy surfaces because of the danger of folliculitis.

8 A pathologic report is important in cases of tumors.

9 Success in treating skin diseases follows the careful and judicious use of medication the actions of which are well known rather than from the application of a multiplicity of drugs.

A healthy condition of the skin is dependent upon the normal functioning of all organs of the body and any deviation from the normal must be corrected. Such disorders as dyspepsia, constipation, menstrual irregularities, anemia, albuminuria, glycosuria, foci of infection etc., must be treated when present.

Patients with acute skin diseases should be confined to bed. The patient should wear garments of cotton or silk and the diet should be easily digestible.

Diet. It has been shown repeatedly experimentally and clinically that the diet plays an important part in the irritability of the skin and in skin diseases in general. For example, the lesions of psoriasis who are heavy drinkers or eat heartily of carbohydrates are much less easily susceptible to therapy. A starvation diet is apt to be of great therapeutic

value to patients with generalized pruritus and inflammatory dermatoses. Certain articles in the diet of patients with acne vulgaris and rosacea definitely prolong and aggravate the condition. In lupus vulgaris, high caloric and high vitamin diets are important and aid the patient's welfare. In certain chronic infectious granulomata such as syphilis, blastomycosis, and actinomycosis, skin conditions develop as a result of an avitaminosis (improper intake, and malabsorption or improper metabolism).

Psoriasis is a disease of the healthy person, especially the well nourished. Reducing such a patient's weight favorably influences the disease.

The effect of diet in food urticarias and food allergies in general is clear cut. Eliminating the offending substances causes the eruption to disappear rapidly or slowly.

A diet consisting of rice or buttermilk only for seventy two hours or longer will often lead to the disappearance of the pruritus of lichen planus and that occasionally observed in psoriasis and some other general dermatoses.

Highly seasoned foods, such as sausages, pickles, condiments, and strong cheese, are avoided. It is advisable to reduce the carbohydrate intake in many cutaneous disturbances, especially in cases of pyoderma.

Patients suffering from seborrhea should avoid white bread, sugar, fatty victuals, and salty foods.

Alcoholic drinks and coffee are avoided while weak tea is allowed. The best beverage for patients with cutaneous disease is pure water taken not less than one hour after or one hour before meals.

In most diseases of the skin a mixed diet is permissible; however, excesses of the fats, carbohydrates, and proteins are avoided. Vegetables and fruits are al-

lowed. Overfeeding is the chief danger in diseases of the skin. Frequently underfeeding will produce a beneficial result, especially in cases of chronic eczema. It is better to eat more frequently than to partake of heavy meals. An occasional fast day during which buttermilk is the sole article of food, is helpful in cases of chronic skin disease.

The question of washing the skin is important. Soaps owe their detergent (cleansing) action to their alkalinity which makes them harmful for certain normal and diseased skins and helpful for others. Some skin diseases, like psoriasis, derive benefit from frequent bathing in warm water and soap. Washing in water alone is not harmful. It is the soap and friction with which it is applied which act as irritants. Acute and weeping eczema is benefited by washing in warm normal salt solution.

Internal Medication. It would not serve any useful purpose to devote a section to the pharmacological action of the many internal remedies useful in the different skin diseases. Some of those in common use which are given orally include:

Arsenic. This is given either as Fowler's solution (potassium arsenite solution) or as arsenic trioxide. It is an alterative, hematinic, and tonic. It is probably the most widely used and abused remedy in dermatology. It is of definite value in the treatment of chronic psoriasis, dermatitis herpetiformis, pompholyx, pemphigus vulgaris, lichen planus, and mycosis fungoides. An excellent mode of administration is as follows:

Fowler's solution (liq. potassii arsenitis) 30.0
T. gratia comp. 60.0

So. For five days, take 5 drops in little water after breakfast. For the succeeding five days, at the same time, take 10 drops. Increase the dose 5 drops at the end of each five-day period, until a maximum of 30 drops is reached.

Then decrease the dose every five days until the 5-drop dose is again reached, extending the treatment over a total of seventy-five days.

The patient should report once weekly for detection of signs of intolerance. If they develop, the dose for that patient is at a maximum and should gradually be reduced. At least two months should elapse before a second cycle of treatment.

Calcium. This is given orally as calcium lactate, calcium gluconate, calcium chlorate, and calcium sulfide. It is useful as a prophylaxis in serum sickness and in the treatment of chilblains, urticaria, and verruca plana. Calcium sulfide is especially indicated in the treatment of boils, abscesses, and pustular acne. Calcium peroxide is used externally in the treatment of infected wounds, while calcium permanganate is useful in the treatment of rodent ulcers and inoperable carcinoma.

Salts of Mercury. These are given orally in such diseases as syphilis and lichen planus. The cyanide of mercury is less irritating than mercuric chloride and is given orally in doses of 4 to 8 mg ($\frac{1}{10}$ to $\frac{1}{8}$ grain).

Dilute Hydrochloric Acid. This is given in the treatment of acne rosacea, pellagra, and allergic skin diseases.

Iodine. In the form of potassium iodide this is useful in the treatment of tertiary syphilis, actinomycosis, blastomycosis, and sporotrichosis.

Sulfonamides. These include, among others, sulfanilamide, sulfapyridine, sulfathiazole, and sulfadiazine. The least toxic as well as the most active of these is sulfadiazine. It is useful in all pyogenic dermatoses as well as some of the virus diseases, especially lymphogranuloma venereum. Its use is indicated in the treatment of erysipelas, erysipeloid, cellulitis, impetigo, furunculosis, and secondary pyogenic infection. Allergic response, toxic manifestations, and fatality

may occur during sulfonamide therapy and one must watch for them. The morbilliform, scarlatiniform and multiform eruptions particularly do not necessarily require stoppage of the drug as they often disappear even though treatment is continued. Before administering any of the sulfonamides urine and complete blood examinations should be made, repeated daily for the first ten days and then every second or third day. Exposure to sunlight or ultraviolet radiation during the administration of the sulfonamides should be avoided as the drug is a photosensitizer and severe dermatitis might develop. The most serious complications of sulfonamide therapy are hepatic damage, acute hemolytic anemia, leukopenia, granulocytosis, hematuria and oliguria.

Thyroid Extract. It is useful in scleroderma, alopecia, acne vulgaris, erythema induratum, some senile eczemas, and ichthyosis. Parathyroid extract, adrenalin and epinephrin are indicated in treatment of acute and chronic urticaria.

Parathyroid Extract. This has also been successfully used in treatment of chilblain and varicose ulcers.

Estron. This is indicated in the treatment of kraurosis vulvae.

Orchitic Extract. This is often helpful in relieving senile pruritus of the male.

Ovarian Hormones. These hormones are used in the treatment of senile pruritus of the female.

Insulin (Extract of Entire Pancreatic Gland). This is occasionally helpful in treating morphea and scleroderma.

Neostigmine Bromide. Given in doses of 15 mg. to 30 mg. three times daily this has given excellent results in the treatment of vitiligo.

Injections. Therapy by injection is employed extensively in dermatology. Appropriate sera are of value in cuta-

neous diphtheria, anthrax, scarlet fever and other streptococcal infections like erysipelas.

Protein shock is used with considerable benefit in chronic diseases of the skin and in desensitizing patients suffering from chronic urticaria and allergic dermatitis. A mild shock may result from an intramuscular injection of 5 to 10 cc. of the patient's own blood into the upper part of the buttock every five days. From 5 to 10 cc. of boiled milk or aolan may be employed in the same manner with similar results. This is useful in chronic infections, such as chronic urticaria, chronic pruritus, and acroas vulgaris.

Intravenous injections of typhoid vaccine in doses of 150,000,000 or more once a week produce marked shock. The subcutaneous injection of this vaccine produces less shock.

Epinephrine chloride or **epinephrine in oil suspension** is useful in urticaria, serum sickness, and angioneurotic edema.

Arsenic may be given by injection. It is prescribed in dermatitis herpetiformis, psoriasis, lichen planus, and pemphigus. Sulzberger and Wolf prefer the subcutaneous administration of a solution of 2 per cent sodium arsenate containing 1 per cent phenol. The initial dose is 1 minim, which is increased 1 minim each day until 30 minims are administered or intolerance results.

Asphenamine, **mapharsen**, **neoarsphenamine**, **sulfarsphenamine** and **tryparsamide** are given intravenously and are antisyphilitic remedies.

Injections of **bismuth** have replaced mercury in syphilotherapy. It is also beneficial in lupus erythematosus and lichen planus.

Injections of **calcium gluconate**, **thio-sulfate** and **calcium chloride** are indicated in acute dermatitis, urticaria, prur-

nitus, pemphigus, and dermatitis herpetiformis.

Intravenous injections of ascorbic acid are useful as a desensitizing agent in allergy.

Intravenous injection of solutions of dextrose are useful in exfoliative dermatitis and lupus erythematosus.

Intramuscular injections of *eneol* (mercury salicylarsenate) are given in lichen planus and psoriasis.

Intravenous and intramuscular injections of mercury cyanide are of definite value in erythema multiforme and lichen planus.

The several salts of gold as well as colloidal gold preparations, either intravenously or locally are of value in chronic discoid lupus erythematosus.

Intramuscular injections of liver extracts are indicated in seborrheic dermatitis, acne vulgaris, pellagra dermatitis exfoliativa, chronic lupus erythematosus, and pemphigus vulgaris.

Intramuscular injections of manganese butyrate are employed in furunculosis and acne vulgaris.

Intramuscular injections of moccasin snake venom are useful in purpura fulminans, recurrent herpes simplex, and for relieving the pain following herpes zoster.

Subcutaneous injections of parathyroid hormone are of value in urticaria, hives, and varicose ulcer.

Intramuscular injections of poison ivy antigen are valuable in prophylaxis against poison ivy oak, and sumac dermatitis. It tends to lessen the duration of attacks when given at their onset.

The substances extracted from poison ivy and poison oak are closely related chemically. According to some observers, ivy and sumac extracts are identical. Ivy antigen has been used for prophylaxis against all three species of *Rhus*.

When injections of ivy extract are used for immunization or treatment frequent small doses are preferable to large doses at long intervals.

Subcutaneous injections of surgical pituitary extract are useful in herpes zoster and acute urticaria.

Subcutaneous injections of sodium chloride are indicated in drug eruptions, especially when the offending agents are the bromides and iodides. It is also helpful in acute dermatitis.

Aqueous solution of calcium sulfadiazine may be administered subcutaneously or intravenously in concentration up to 4 per cent with no ill effects. The pattern of absorption and excretion of calcium sulfadiazine as measured by the rise and fall of drug level in the blood, does not differ significantly from that observed following the parenteral administration of comparable doses of sodium sulfadiazine. Clinical experience indicates that calcium sulfadiazine administered subcutaneously is effective in establishing and maintaining adequate blood levels of the drug in patients requiring parenteral chemotherapy.

Aqueous solutions of calcium salts of thiazole administered subcutaneously has resulted in local inflammation.

It has been reported that solutions of sodium sulfadiazine in concentrations up to 8 per cent may be safely administered subcutaneously.

Penicillin. This and other antibiotics (streptomycin, tyrothricin, and streptothricin) are potent antibacterial agents. Penicillin is produced by a mold, the *Penicillium notatum*. It may be used intravenously, intramuscularly, or topically in ointment, in isotonic saline solution, 3 per cent dextrose solution, preferably sterile distilled water or suspended in peanut oil and beeswax. For intravenous use a concentration of 100 units

per cc. is used for intramuscular use, a concentration of 50 000 units per cc. It is less effective if given orally and must be buffered to offset the destructive action of the gastric secretions. Single intramuscular injections of penicillin in peanut oil and beeswax (1 cc. containing 300 000 units) at twelve hour intervals, provide a continuous high penicillin blood serum concentration. Penicillin is indicated in the treatment of a variety of infections due to the hemolytic streptococcus, the staphylococcus, the pneumococcus, *Treponema pallidum* and the gonococcus. Streptomycin is useful in tularemia.

Tyrothricin is used only in the form of a topical application to infected areas.

Intravenous injections of *sodium thio-sulfate* are beneficial in chronic eczema, chronic urticaria and dermatitis exfoliativa resulting from bismuth, mercury and arsenic therapy.

Subcutaneous perianal injections of *absolute alcohol* are beneficial in chronic resistant pruritus ani. Infiltration of the perianal region with *isotonic salt solution* is at times equally effective.

Subcutaneous injections of *testicular hormones* have been reported to effect recovery in tinea tonsurans of the male child.

The *sodium salts of sulfathiazole* or *sulfadiazine* may be administered intravenously in 5 per cent concentration. As an initial dose, 4 gm. may be given then 1 gm. every six hours.

Vaccines. Suspensions of killed bacteria and/or their products are used as bacterins or vaccines in a variety of infectious dermatoses. They are given subcutaneously or intravenously. The material may be a stock vaccine consisting of a mixture of various organisms, or an autogenous vaccine prepared from or

ganions cultured from the lesion. The action of vaccines is that of a foreign protein. Autogenous vaccines are occasionally serviceable in chronic urticaria, furunculosis, and pyoderma. Vaccine of colon bacillus is indicated in pruritus ani. Streptococcus vaccine is valuable in streptococcal dermatitis, cellulitis, and erysipelas. The work of Bestedka led to the use of antiviral, toxoid antiserum, bacteriophage etc. for the treatment of local cutaneous infections due to staphylococci and streptococci. These preparations are occasionally successful, however the advent of the sulfonamides has largely displaced this form of therapy.

Local Medication. Smith (Herbert) outlines the usual drugs used locally in dermatology as follows:

(1) *Bland Oil and Ointment Bases*

Oleum olivae
Oleum gomypii seminis
Oleum lini
Oleum amygdalae expressum
Oleum ricini
Oleum maydis
Oleum sesami
Oleum morrhuae
Oleum theobromatis
Petrolatum liquidum
Adeps benzoïnatus
Unguentum
Ceratum
Unguentum albei cordi
Unguentum aquae rosae
Petrolatum
Petrolatum album
Adeps lanae
Adeps lanae hydrosus
 Other cholesterol derivatives.

(2) *Aqueous Lotions*

Witch hazel and lime water
 Isotonic salt solution
 Boric acid solution
 Resorcin and boric acid solution
 Sodium thiosulfate solution
 Aluminum acetate solution (Barrow's Solution)
 Potassium permanganate solution
 Lead water

Solution of iron chloride (may cause pigmentation)

Liquor calci sulfurati (Vincetack Lotion)

Liquor methylrosanilini (gentian violet 3 per cent)

(3) *Protective Powders:*

Zinc oxidum

Zinc carbonas precipitatus

Zinc stearas

Neosalicinas preparata

Bismuthi subcarbonas

Bismuthi subnitras

Magnesi carbonas

Bentonite (colloidal clay forms gel with water; oils and fats emulsify easily with this gel)

Talcum purificatum

Terra silicea purificata

Amrylitas

Lycopodium

Acidum boricum.

(4) *Facilitating and Greasiness Creams.*

(5) *Aqueous Lotions Containing Insoluble Powders:*

Neosalicinas lotion

Neosalicinas lotion phenolated

Loto alba

Loto nigra

Loto Flava

(6) *Lotions:*

Limeastrum calcis

Limeastrum neosalicinas

Alcohol with castor oil as vehicle.

(7) *Local Sedatives:*

Menthol

Phenol

Cresosol

Camphor and phenol

Camphor and chloral

Liquor creosoti suspension

Betula (3 per cent)

(8) *Stimulants, Antipruritics, and Astringents:*

Hydroxyannus ammoniacum

H₂ drangyrum chloridum corosivum

H₂ drangyrum chloridum alkali

H₂ drangyrum oxidum flavum

Unguentum hy drangyri

Unguentum hy drangyri dilatum

Unguentum hy drangyri alutale

Oleatum hy drangyri

Iodum

Thymolis iodidum

Sulfur sublimatum

Sulfur precipitatum

Resorcinol

Betasaphthol

Chlor-hydroxy-quinoline elastum (Quinolox)

Chrysarobinum

Neosalin

Dihydroxy anthracenol (anthralin, 1, 9, 9, 1, 1 per cent elastum)

Sulfonamides in elastum or suspension (3.20 per cent)

Penicillin elastums (1000 units to gram)

(9) *Parasitocides and Fungicides:*

Acidum salicylicum

Acidum benzoicum

Balsamum peruvianum

Thymolis

Oleum caryophylli

Oleum cassiae

Fix pine

Fix juniperi

Fix carbonis

Oleum picis rectificatum

Naftalen

Icthoanthracol.

(10) *Greasiness Creams Emulsifiers Emulsions*

Surface Active Agents Wetting Agents

Synthetic Detergents Water Soluble Oil

and Elastum Bases.

These are, or contain, substances which alter surface tension of solvents. Recently the usefulness of preferential wetting in a variety of pharmaceuticals has been widely recognized. The value of many of these surface active agents as a real aid in dermatologic therapy is constantly being investigated, although some have already proven their worth in certain skin conditions noted in the text.

Among surface active agents are:

Glycerol monostearat

Carbowaxes (polyethylene glycol compounds)

Hydrophilic elastum, U.S.P. XIII

Hydrophilic petrolatum, U.S.P. XIII

Sulfated hydrogenated castor oil N.P.

Deponal (sodium lauryl sulfate)

Stanol (stearyl alcohol)

Cetyl alcohol

Tricethanolamine

Quaternary ammonium chlorides, such as pyrazine 1002

Pectin

Tweens and spans

Sulfonates, lathering and nonlathering soapless detergents or soap substitutes.

There is a normally high degree of impermeability to the skin. It has been demonstrated that use of surface active agents in the compounding of topical applications has influenced this permeability to a great degree. Drugs dissolved in "wetted" water or solutions render the skin completely permeable to many of these substances. It should therefore be remembered that the continuous use of such cleansers or soap substitutes for example may be harmful. When using such a soap substitute ordinary soap must be avoided or irritation due to them frequently occurs. Furthermore patients may become sensitive to wetting agents. In certain oily states, acne vulgaris in particular these soap substitutes tend to cut sebaceous films on the skin to a greater degree than ordinary soap and are helpful. Dry skins and those sensitive to ordinary soaps may be irritated by them. In most cases, superfatted soaps (Baus Althene or Shulton's lanolin soaps) are preferable when soap substitutes are needed. However creams with both detergent and emollient properties, which will not dry the average skin have been developed and widely used (pHsoderm). pHsoderm is active under acid alkaline and neutral conditions and when used with hard soft hot, cold or sea water.

Triethanolamine is also extensively used in the manufacture of cosmetics, soap, and cosmetic creams, and to some extent in dermatologic therapy. It is said to have emulsifying and skin softening properties when used in ointments and oily bases.

Carbowax compounds among the newer ointment bases, are water soluble polyethylene glycols with fat or waxlike consistency. Although they stick to moist surfaces, they can easily be washed off with warm water.

Application of medicinal substances is of value in the protection they afford. The form of local preparation in use is selected according to the following plan.

1 Powders and lotions are indicated in simple erythema, urticaria, edema and subacute dermatitis.

2 Lotions and liniments are of benefit in cutaneous diseases in which there is a marked acute inflammation and weeping.

3 Oils, ointments, and starch poultices are useful in dry, scaly and crusty cutaneous lesions.

4 Hot fomentations, baths, and starch poultices are indicated in treating septic lesions.

5 Papular lesions are best treated by lotions, ointments and pastes.

6 Pastes are advised for subacute stages of eczema and dermatitis.

7 Ointments are more helpful in psoriasis and lichen planus.

The general rule in local medication is to apply the absolute minimum of the medicament as a dressing. Thick layers of lint and cotton are avoided. Pieces of linen or white rayon are employed as a protective dressing whenever the patient is up and about.

Baths. The question of bathing is very important in dermatology.

It is necessary to cleanse the skin of cutaneous excretions in order to keep it active and healthy. A normal skin sheds dead epidermal cells in the form of fine scales. Sweat and sebaceous secretions form an unpleasant coating on the skin when incorporated with these epidermal scales. As a general rule bathing is contraindicated in acute inflammatory disease of the skin, although a weeping eczema may occasionally be benefited by local washing in warm water or normal salt solution.

Ordinary hot water baths are useful in many dry forms of skin diseases like psoriasis, xeroderma, ichthyosis, etc. The average time of immersion in a water bath is about ten minutes at a temperature of 103° F (41° C). The average bath contains 115 liters (25 gallons).

The bath is improved by the addition of agents which are employed for their soothing, astringent, antiseptic and stimulating action. The type of medication employed is indicated by the cutaneous disease present.

Soothing and Emollient Baths Oat meal, bran, malt, or laundry starch soften hard water and render it soothing. These baths are indicated in all acute exudative and pruritic dermatoses which include plant dermatitis, urticaria, pityriasis rosea, acute lichen planus, etc. Oatmeal, bran, and malt baths are made by placing about 4 pounds of any of these ingredients in a cheesecloth or muslin bag which is suspended directly under the hot water tap so that its demulcent constituents are carried into the bath. The resulting sedative bath is rendered antiseptic by the addition of 2 ounces of boric acid powder.

The starch bath is made by incorporating a pound of starch in cold water to make a paste which is then added to the hot water bath.

An *alkaline bath* is soothing to inflamed surfaces. It is prepared by adding 8 ounces of sodium bicarbonate or 6 ounces of potassium carbonate to 30 gallons of water at a temperature of 105° F.

A *borax bath* is made by adding 3 ounces of borax to a bath of 30 gallons of water at a temperature of 105° F.

An *acid bath* is made by adding an ounce of nitric or hydrochloric acid or 10 ounces of vinegar to 30 gallons of water in a porcelain bath. Acid baths are indi-

cated in chronic lichen prurigo and urticaria.

Antiseptic baths are made by adding 4 ounces of zinc sulfate to 30 gallons of water. Potassium permanganate ($\frac{1}{2}$ dram to 2 gallons) or a mercurial bath (2 drams hydrochloric acid, 1 dram of biniodide of mercury and 2 ounces of sodium chloride in 30 gallons of water) makes an agreeable antiseptic bath. The mercurial bath is useful in infantile syphilis and widespread syphilodermatous.

An *iodine bath* is made by adding 1 dram (4.0 gm) of iodine crystals and 2 ounces (60 cc) of liquor potassae to 30 gallons of water. This bath is indicated in furunculosis, Darier's disease, pemphigus, and granuloma inguinale.

Saline baths are useful in extensive sepsis, dermatitis, and lurns. These baths are made slightly hypertonic by adding 3 pounds of common salt to 30 gallons of water at a temperature of 100° F.

A *sulfur bath* may be made by adding 3 ounces of potassium sulfate to 30 gallons of hot water. Colloidal sulfur (20 gm (4 ounces)) makes an excellent bath. Viemick's solution (100 gm calcium sulfate) 150 to 200 gm added to 30 gallons of water is another worthwhile sulfur bath. The duration of a sulfur bath should not exceed twenty minutes. The sulfur bath is recommended for scabies, mycotic dermatoses, multiple furunculosis, impetigo and staphylococcal dermatitis.

Astringent Baths Oak bark is a useful astringent for pruritus, eczema and the pruriginous affections of children. An oak bark bath is prepared by boiling 3 pounds of oak bark in 6 liters of water which are added to 30 gallons of water. The oak bark may be substituted by 300 gm. of tannin.

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III

Thymol	1.0
Phenolic liquid	1.0
Neo-calamine prep.	25.0
Borax	7.6
Euc phenomol	10.0
Glycerin	10.0
Liq. potassae	8.0
Aq. dist.	q. s. 250.0

An antipruritic lotion indicated in formic acid, folliculitis or their vesicles

A saturated solution of boric acid or a 1 per cent solution of resorcin in aqua hamamelidis is a good antiseptic lotion.

The well-known calamine lotion is an astringent combining the properties of a powder and a lotion

Calamine perf.	8.0
Calcium carbonate	8.8
Liq. phenol subacetate dil.	8.0
Glycerin	8.0
Aq. calid.	q. ad. 100.0

The evaporation of water in calamine lotion cools the skin; the lead water is soothing and astringent, the calamine and powdered zinc oxide act as a protective covering; the glycerin makes them adhere.

Small amounts of glycerin (3 to 10 per cent) are frequently added to lotions to prevent complete drying as well as to keep the irritated skin soft and pliable. However large amounts must be avoided, since glycerin is hygroscopic undiluted, it exerts a drying action harmful to an already irritative or sensitive skin surface.

A ample softening or emollient lotion, to prevent chafing or for skins sensitive to soap, is as follows

Boric acid	3.6
Glycerin	10.0
Aq.	250.0
Pal. tragacanth	1.2
Oil rose	8.8

Lotions are shaken well before use, poured in a saucer applied to the affected parts with a camel's hair brush

or a piece of lint and left to dry. They may however be applied as a wet dressing. Lotions may be thickened by adding a 5 per cent solution of bentonite.

Wet Dressings Wet dressings are applied on raw surfaces once every three or four hours. These wet dressings are of two kinds.

1. Wet dressings applied under impermeable cover. This consists of ten or more layers of muslin, linen, or rayon saturated in the prescribed medication and protected with an impermeable cover like oil silk. An electric pad or hot water bag may be placed over the dressing to keep it warm. A dressing of this kind is renewed every four hours. This type of dressing is indicated in deep-seated inflammatory processes of an infectious nature and in chronic dermatoses.

2. The open wet dressing is extensively used in dermatology. It is indicated in acute cutaneous inflammatory conditions, especially whenever oozing is present. It is also useful in secreting genital and intertriginous eczema. The action of the open wet dressing is antiphlogistic and antipruritic. It also functions as a cleansing agent by absorbing the drainage.

The wet dressing is employed according to the following plan: (1) Soak several pieces of muslin, linen, or white rayon in the desired medicament at room temperature. (2) Apply soaked dressing as a compress and renew every five minutes. The dressing remains uncovered and is not allowed to dry. Rayon is less likely to stick to the tissues than gauze. Gauze previously soaked in paraffin and wrung out before application of the medicament will be less likely to stick to the raw surface.

The following preparations comprise the most widely used solutions for wet dressings:

A *tar bath* consists of 3 ounces of liquor carbonis detergens or liquor picis carbonis in 30 gallons of water. Tar baths are indicated in pruritus, chronic eczema, and universal psoriasis.

A *cedar bath* is prepared by adding the following preparation to 30 gallons of water:

Ol white birch	100.0
Tr saponis molle	75.0
Aq	75.0

The *continuous bath* is useful in pemphigus, severe burns, decubitus, dermatitis exfoliativa and gangrene. The question arises as to keeping the water at an even temperature of 37° C since the patient may have to be kept in the bath for days or weeks. The continuous bath is not always available. In the absence of this bath the patient is placed on a stretcher which is so arranged as to permit raising and lowering in the bath thus keeping the patient floating. The bath is started at a temperature of 32° C and gradually raised and held at 37° C. Boards and blankets are placed over the tub to prevent cooling. A leather strap is suspended from the ceiling to enable the patient to adjust his position at will.

A variety of vapor or hot air baths are available.

The *radiant heat baths* are dry and consist of passing the electric current through wires or by employing electric light bulbs.

The simplest *vapor bath* is the ordinary steam bath or the Russian bath. The *Turkish bath* is essentially a dry hot air bath in which moisture is produced by sweating.

Medicaments like sulfur and oatmeal may be added to these baths. Sulfur and calomel are volatilized in this case by heat. Sulfur is useful in scabies while calomel is beneficial in syphilis.

Soaps Soaps have limited applications in dermatology. Ordinary toilet soap and soft soap (sapo viridis) are useful in acne vulgaris for removing sebum. Laundry soaps are alkaline and are, therefore, useful in scabies, psoriasis, and chronic seborrheic dermatitis. Soft soap is ideal for the removal of crusts and of considerable value in xeroderma and ichthyosis. Patients with dry skin are advised to employ superfatted soap like basis soap, hazeline soap or Erchoff's superfatted soap. Medicated soaps are not effective unless the lather remains on the skin or unless it is protected with an impervious material like oil silk or rubber. This may however lead to dermatitis venenata. Their usefulness is therefore limited. Medicated soaps contain sulfur, tar, mercury, ichthyol, balsam of Peru, etc.

Sulfonated soaps are nonlathering oily liquid detergents. The widely used sulfonated soaps or soap substitutes (Drene, Acidolate, and Tensol) are water soluble and may or may not be irritating (see surface active agents, p. 15).

Lotions Lotions are largely used in dermatology as cooling, astringent, antiseptic, and antipruritic agents. They are aqueous solutions containing soluble or insoluble drugs.

The following preparations are examples of soothing lotions.

I	
Resorcin	5.0
Liquor plumbi subacetatis fort	5.0
Aq camphorae	60.0
Aq dist.	q.s. 500.0

II	
Zinc sulfocarbolate	2.0
Pulv zinc oxid	6.0
Calcium carb.	8.0
Glycerin	6.0
Aq calcis	q.s. 100.0

See. Indicated for acute pruritic dermatitis.

III

Thynol.	1.0
Phenol. liquidacti	III
Neo-calamine prep.	35.0
Bentonite	7.0
Zinc phenolif. cont.	10.0
Glycerin	10.0
Liq. potassae	2.0
Aq. dest.	q. s. 250.0

An astringent lotion indicated in furunculosis, folliculitis or tinea cruris.

A saturated solution of boric acid or a 1 per cent solution of resorcin in aqua hamamelidis is a good antiseptic lotion.

The well-known calamine lotion is an astringent combining the properties of a powder and a lotion.

Calamine powr	60
Calcium carbonate	60
Liq. plumbi subacetatis dil.	60
Glycerin	6.0
Aq. calcis	q. s. ad. 100.0

The evaporation of water in calamine lotion cools the skin, the lead water is soothing and astringent, the calamine and powdered zinc oxide act as a protective covering; the glycerin makes them adhere.

Small amounts of glycerina (3 to 10 per cent) are frequently added to lotions to prevent complete drying, as well as to keep the irritated skin soft and pliable. However large amounts must be avoided, since glycerin is hygroscopic; undiluted, it exerts a drying action harmful to an already irritative or sensitive skin surface.

A simple softening or emollient lotion, to prevent chafing or for skins sensitive to soap, is as follows:

Boric acid.	36
Glycerin	100
Lq.	200
Pal. turgensib.	12
Ol. roseae	63

Lotions are shaken well before use, poured in a saucer, applied to the affected parts with a camel's hair brush

or a piece of lint and left to dry. They may however be applied as a wet dressing. Lotions may be thickened by adding a 5 per cent solution of bentonite.

Wet Dressings. Wet dressings are applied on raw surfaces once every three or four hours. These wet dressings are of two kinds:

1. Wet dressings applied under impermeable cover. This consists of ten or more layers of muslin, linen, or rayon saturated in the prescribed medication and protected with an impermeable cover like oil silk. An electric pad or hot water bag may be placed over the dressing to keep it warm. A dressing of this kind is renewed every four hours. This type of dressing is indicated in deep-seated inflammatory processes of an infectious nature and in chronic dermatoses.

2. The open wet dressing is extensively used in dermatology. It is indicated in acute cutaneous inflammatory conditions, especially whenever oozing is present. It is also useful in secreting genital and intertriginous eczema. The action of the open wet dressing is antiphlogistic and antipruritic. It also functions as a cleansing agent by absorbing the drainage.

The wet dressing is employed according to the following plan: (1) Soak several pieces of muslin, linen, or white rayon in the desired medicament at room temperature. (2) Apply soaked dressing as a compress and renew every five minutes. The dressing remains uncovered and is not allowed to dry. Rayon is less likely to stick to the tissues than gauze. Gauze previously soaked in paraffin and wrung out before application of the medicament will be less likely to stick to the raw surface.

The following preparations comprise the most widely used solutions for wet dressings.

A *tar bath* consists of 3 ounces of liquor carbonis detergens or liquor picis carbonis in 30 gallons of water. Tar baths are indicated in pruritus, chronic eczema, and universal psoriasis.

A *cedar bath* is prepared by adding the following preparation to 30 gallons of water:

Ol. white birch	100.0
Tr. saponis mollis	75.0
Aq.	75.0

The *continuous bath* is useful in pemphigus, severe burns, decubitus, dermatitis exfoliativa, and gangrene. The question arises as to keeping the water at an even temperature of 37° C since the patient may have to be kept in the bath for days or weeks. The continuous bath is not always available. In the absence of this bath the patient is placed on a stretcher which is so arranged as to permit raising and lowering in the bath thus keeping the patient floating. The bath is started at a temperature of 32° C and gradually raised and held at 37° C. Boards and blankets are placed over the tub to prevent cooling. A leather strap is suspended from the ceiling to enable the patient to adjust his position at will.

A variety of vapor or hot air baths are available.

The *radiant heat baths* are dry and consist of passing the electric current through wires or by employing electric light bulbs.

The simplest *vapor bath* is the ordinary steam bath or the Russian bath. The *Turkish bath* is essentially a dry hot air bath in which moisture is produced by sweating.

Medicaments like sulfur and oatmeal may be added to these baths. Sulfur and calomel are volatilized in this case by heat. Sulfur is useful in scabies while calomel is beneficial in syphilis.

Soaps Soaps have limited applications in dermatology. Ordinary toilet soap and soft soap (sapo viridis) are useful in acne vulgaris for removing sebum. Laundry soaps are alkaline and are, therefore, useful in scabies, psoriasis, and chronic seborrheic dermatitis. Soft soap is ideal for the removal of crusts and of considerable value in xeroderma and ichthyosis. Patients with dry skin are advised to employ superfatted soap like basis soap, hazelme soap or Eich-off's superfatted soap. Medicated soaps are not effective unless the lather remains on the skin or unless it is protected with an impervious material like oil silk or rubber. This may however lead to dermatitis venenata. Their usefulness is therefore limited. Medicated soaps contain sulfur, tar, mercury, x-thyol balsam of Peru, etc.

Sulfonated soaps are nonlathering oily liquid detergents. The widely used sulfonated soaps or soap substitutes (Drene, Acidolate, and Tensur) are water-soluble and may or may not be irritating (see surface active agents, p. 15).

Lotions Lotions are largely used in dermatology as cooling, astringent, antiseptic, and antipruritic agents. They are aqueous solutions containing soluble or insoluble drugs.

The following preparations are examples of soothing lotions.

I	
Resorcin	5.0
Liquor plumbi subacetatis fort.	5.0
Aq. camphorae	80.0
Aq. dist.	q.s. 100.0

II	
Zinc sulfocarbolate	5.0
Polv. zinci oxidii	0.5
Calcium carb.	5.0
Glycerin	0.0
Aq. caloris	q.s. 100.0

8cc. Indicated for acute pruritic dermatitis.

Equal parts of boric acid, tannic acid and subgallate of iron form an excellent dry powder for hyperdermatitis.

Equal parts of calomel and talcum make a good remedy for erosive balanitis.

The liberal dusting of powder is a valuable aid for a number of pruritic conditions, erythematous lesions, early stages of eczema, and first-degree burns. Powders are contraindicated in profuse oozing dermatitis or on any lesion which is purulent.

Ointments Ointments consist of a fatty base with one or more medicinal agents. The common bases are benzoinated lard, yellow petrolatum, lanolin, aquaphor mixtures of hard and soft paraffin, and mixtures of lanolin and petrolatum. Petrolatum is the simplest base but it is very impervious to perspiration to have wide applications. Aquaphor (cholesterolized petrolatum) is one of the best bases because it is insoluble in water and soluble in ether and chloroform. Aquaphor combines with about three times its weight in water thus forming a white stable emulsion.

White petrolatum is irritating to some skins on account of the carbon disulfide employed in its manufacture. The yellow petrolatum is therefore a better form.

Ointments are not suitable for oozing or inflamed areas because fats do not mix with serum and they interfere with heat radiation.

Glycerite of starch is a nongreasy base which can be readily removed by water.

Equal parts of cetyl alcohol, petrolatum and mineral oil make a very satisfactory ointment base for the scalp because it is not greasy and drugs can be readily incorporated in it.

Lotions and wet dressings are no longer helpful when the acute stage of

dermatitis subsides. An ointment is then beneficial. The most widely used ointments in America are ung. acid. boric, ung. zinc oxide and ung. hydr. amm. cp (N.F.). The following ointment is more useful.

Cinnabaris	10
Pulv. (radicalis)	100
Petrolatum (yellow)	q. ad 1000
Also useful in impetigo contagiosa, folliculitis, furunculosis, etc.	

Cold Creams A cold cream is a thin ointment consisting of a highly hygroscopic base and containing from 20 to 50 per cent of water. Cold creams are applied thickly. They are well tolerated by the inflamed skin and relieve congestion and pruritus. Their action is soothing and cooling as a result of the evaporation of their water content.

Very helpful in treating acne vulgaris is the following:

Pulv. zinc oxide	10
Greenish starch	10
Lac. salicyl.	10
Salicyl. sulfuric	10
Chloroform	10
Greenish cream	q. s. 300

Use Apply 4 or 5 daily preceded by lot application of boric acid solution.

Oils Some vegetable fats and hydrocarbons are useful for removing crusts and scales, and act as valuable additions to lotions and ointments.

Some patients are allergic to vegetable oils, hence the use of mineral oil is preferable for the removal of ointment before making new applications. Mineral oil is also an unfavorable culture medium for micro-organisms, hence there is preference for its universal use. The most widely used oils are mineral oil, olive oil, linseed oil, castor oil, cod liver oil, oil of sesame, and almond oil.

Nutritive oils like cod liver oil can be irradiated and are in this form useful in ichthyosis and hypovitaminosis A.

1 *Boric acid* (2 to 3 per cent) is indicated in acute or weeping lesions. It does not precipitate the albumen of secretion.

2 *Liquor plumbi subacetatis dilutus* (2 per cent) is mildly astringent and antiseptic. This solution is not to be employed near the eyes.

3 *Resorcin* is prescribed in solutions of from 1 to 3 per cent. It is useful in pyogenic affection.

4 *Acetate of aluminum* is employed in an aqueous solution of from 0.5 to 2 per cent. It is a mild astringent and antiseptic. Aluminum acetate tends to precipitate albumen and should therefore not be employed on oozing surfaces because of its irritating nature.

5 *Tannic acid* (1 to 3 per cent) or *tannin* (1 to 2 per cent) is advised in an aqueous solution. They are astringents and tend to precipitate the albumin in secretions. Tannic acid and tannin are indicated in tinea versicolor, eczema, and superficial edema.

6 *Silver nitrate* (0.07 to 0.5 per cent), *argyrol* (5 per cent) and *protargol* (3 per cent) are useful as astringents and stimulants of granulations. Silver nitrate solution (1:500) is especially useful in necrotic ulcers. Its efficacy is increased by adding 20 per cent alcohol.

7 Moist dressings of the following are indicated for digesting cicatrices, keloids, contractions from burns, etc.

<i>Pepsin</i>	10.0
<i>Hydrochloric acid</i>	1.0
<i>Phenol</i>	1.0
<i>Aq. dist.</i>	q. s. 200.0

A solution of trypsin consisting of the following may be similarly employed.

<i>Trypsin solution</i>	2.0
<i>Sod. carbonate</i>	1.0
<i>Aq. dist.</i>	q. s. 200.0

8. Apply under an occlusive dressing of oiled silk or rubber.

Ahlsvede suggests that keloids be softened with 10 per cent pyrogallol before using either of the above lotions.

8 Dressings of alcohol are useful in lymphangitis, the involution of the abscesses, and in furuncles. Schaeffer suggests the following alcoholic solution.

<i>Acid boric</i>	2.0
<i>Spt. vin. rect.</i>	25 to 75 per cent . q. s. 200.0

The compresses are covered with oil silk or rubber. The impermeable covering is perforated whenever the alcohol is concentrated. Compresses are renewed every five hours.

Powders. Powders dry the skin, cool it and prevent friction of adjacent surfaces. They are indicated in pruritic, erythematous, and edematous lesions.

Powders are of mineral or vegetable origin. The chief mineral powders in dermatology are zinc oxide, zinc stearate, insoluble salts of bismuth, talcum, titanium oxide and kaolin. The main vegetable powders are starch and lycopodium.

Vegetable powders are more soothing than mineral powders but have the disadvantage of fermenting and decomposing in the presence of moisture. The following is a mild antiseptic powder.

<i>Acid boric</i>	2.5
<i>Pul. zinc oxid.</i>	20.0
<i>Pul. talc</i>	20.0

An antiseptic powder for drying moist surfaces is prepared as follows.

<i>Bismuth subnit.</i>	30.0
<i>Talc</i>	60.0
<i>Zinc oxid.</i>	10.0

Sulfanilamide and sulfathiazole may be employed in powdered form and are indicated in local pyogenic infection. However sensitization often occurs.

A mild antipruritic powder consists of

<i>Menthol</i>	25
<i>Zinc oxid.</i>	5.0
<i>Talc</i>	5.0

Antipruritics Antipruritics are important medicaments which include menthol, phenol, thymol, camphor lysol, liquor carbonis detergens, nupercaline, benzocaine, procaine, butyn, diathane hydrochloride, halocaine, nupercaline, witch hazel, alcohol, hot or ice water cornstarch, oatmeal, sodium bicarbonate, and vinegar. Three factors are considered in deciding upon antipruritic preparation: (1) The drug to be used; (2) the strength of application, and (3) the type of suitable vehicle, whether lotion, ointment, paste, etc.

It is necessary to remember when selecting the drug to be used, that in many infectious conditions like ring worm of the feet and impetigo the skin is very intolerant to substances ordinarily not irritating. It is, therefore, a better therapy to start with soothing remedies until acute stages have elapsed, then gradually add the desired antiseptic.

The skin of patients varies in the tolerance of the individual to drugs. It is prudent to begin with a weak concentration of medicament and gradually increase the drug whenever necessary.

The type of preparation is decided on the following plan:

Erythema, wheals, edema, and vesicles call for powders and lotions.

Weeping surfaces require lotions, liniments, and creams.

Crusts demand oils, ointments, and starch pastilles.

Scales necessitate ointments.

Papular lesions call for lotions, ointments, and pastes.

Subacute stages of eczema and dermatitis require pastes.

Pruritus demands powder lotions, and ointments.

Ointments and pastes are applied upon gauze or muslin held in place with a light bandage.

Silk and rayon are useful because of their nonadhering quality.

Physiotherapy Physiotherapy has numerous applications in dermatology. It includes the following agents: (1) Refrigeration by employing solid carbon dioxide; (2) heat by galvanocautery and diathermy; (3) electricity by employing galvanic baths, electrolysis, and ionization, and (4) radiation by applying ultra violet light, roentgen rays, grenz rays, and radium.

Refrigeration. Refrigeration by solid carbon dioxide, or cryotherapy, is of considerable service for nevi, warts, moles, acne vulgaris, patches of lupus erythematosus, lupus vulgaris, superficial rodent ulcer, and larva migrans. Snow is obtained by allowing the carbon dioxide gas to escape from the cylinder in which it is contained under great pressure. The snow which is thus produced is compressed into a pencil, using a commercial mold sold for this purpose, or an ear speculum or tube made of blotting paper covered with chamois skin. The desired shape and size of snow is molded by pressure against a metal surface or by means of a commercial mold to make its application convenient over the lesion. The snow may also be readily incorporated into soft slush with acetone to make it possible to paint lesions. The period of applying refrigeration varies with the purpose for which it is applied. From twenty seconds to two minutes is usually sufficient. The pressure of application varies in the same way. Firm pressure for ninety seconds is sufficient for deep refrigeration in cavernous nevus. Firm pressure for two minutes is indicated in rodent ulcer and lupus vulgaris. Pressure for twenty seconds is sufficient for lupus erythematosus.

A large blister is formed following refrigeration by carbon dioxide snow.

Liniments Liniments are oily lotions indicated for acute dermatitis and burns. They are applied thickly and lightly covered with gauze, or applied to muslin. Liniments have the cooling effect of calamine lotion and do not undergo the drying characteristic of calamine lotion. The following are among the many useful liniments.

Zinci oxidi	8.0
Ung. aquaphor	8.0
Ol. olivae	30.0
Aq. calcei	q.s. 30.0

Str. Apply every four hours.

II

Ol. olivae	100.0
Oleic acid	25.0
Triethanolamine	4.0
Aq.	q.s. ad. 380.0

M. Add oleic acid to oil, shake well, then add triethanolamine and finally the water.

Pastes Pastes are preparations containing solid ingredients and an oily base of about equal parts. Pastes are less healing and produce a less degree of maceration of the epidermis. Pastes form a more protective covering than ointments and absorb exudates. The commonly used paste is the so-called *Laszars paste*.

Zinci oxidi	8.0
Pulv. amyli	8.0
Petrolati	16.0

Paints and Varnishes Paints and varnishes are less effective than ointments. The bases employed for paints and varnishes are collodion, traumaticin (40), chloroform (50.0), and Unna's zinc gelatin.

Unna's zinc gelatin

Gelatin. alba	15.0
Glycerini	15.0
Pulv. zinci oxidi	30.0
Aq.	50.0

Sol. Useful as a supportive dressing for wet.

Peck's varnish (tragacanth 6.0, glycerin, 2.0, water 92.0) is useful in incorporating various medicaments with it.

Ichthyol varnish consists of ichthyol, camphor, charcoal, and tragacanth. It is indicated in cellulitis, erysipelas, and lymphangitis.

Paraformaldehyde powder (Merck) (1.25) in collodion (4.0) is excellent for removing warts. Warts are painted three times each week.

Plasters Plasters are the most penetrating form of applying medicaments. They are not porous and are impervious to secretions. The contraindications are hyperacute and oozing lesions. Good plasters contain a mixture of caoutchouc which is stiffened by a layer of gutta percha and muslin. The plaster of universal application is the salicylic plaster. It is indicated for corn callus, hyperkeratotic and chronic noninflammatory lesions, and is obtainable in strengths varying from 10 to 40 per cent.

The following is useful in alopecia areata, small areas of psoriasis, and small mycotic lesions.

Chrysarobin	5.0
Lanolin	5.0
Wax	2.0

Salve Pencils These have limited applications and of these camphor ice is a common example. The base of these pencils consists of lanolin 2.0 and wax, 1.0. The following preparation is indicated when a softer pencil is desirable.

Cocoa butter	2.0
Wax	1.0
Lanolin	1.0

Use as base for soft pencils.

The following pencil is useful in alopecia areata and small mycotic lesions.

Acid pyrogallie	15.0
Acid salicylic	15.0
Lanolin and wax base	70.0

it through a sheet of paper to a cake of dry soap held in the hand. Where the spark strikes the soap, a dry powder from the dehydrated soap will be found and the paper not marked. A longer spark will cause more rapid destruction, but unnecessary increase of amperage through the rheostat incurs the risk of charring effects. A rapid burning effect is highly undesirable.

The best results follow when the spark is so cool that a little time is required to secure the desired devitalization. The heat is most intense where the spark strikes and diminishes in its tissues as the distance from this point increases. The time factor permits better heat penetration to regions adjoining the growth thus often devitalizing abnormal cells while more resistant normal structures survive.

Often the shrinkage from dehydration is so marked that the adjacent skin is wrinkled as if drawn in by a purse-string suture; and, surprisingly the resultant mark will usually be smaller than the area occupied by an original vascular growth, for example.

Small growths may often be treated without local anesthesia. Intermittent touches of the spark and the small amount of destruction at each application rendering the proceeding tolerable. Local anesthesia should be at some depth as the heat extends beyond the actual destruction.

Except with small growths, a guiding mark should be made around the edge with a very small spark, so that if shrinkage occurs the treatment may be kept within the outlined area. After desiccation, a light touch of a curet will often remove the growth en masse. Enthusiastic curetage in any benign condition adds unnecessary injury. Except in epitheliomata, it is usually best to stop at

the skin level to avoid depressed scars. If one has been too conservative subsequent treatment incurs no risk of missing a perfect result.

Small pedunculated growths may be painlessly removed by grasping the bases with a fine forceps and applying the spark to the growths. After removal, a touch at the base is usually desirable.

The needle is inserted in a growth of firm consistency. A jump spark in such a case will spear off into the adjoining more vascular and better conducting tissues. A luminous appearance within the growth is an indication that the region illuminated is devitalized.

Superficial blemishes that can be hidden by cosmetics present a problem. A scar may arouse more suspicion than the port wine mark which it displaces.

If the surface remains dry after treatment no dressing may be needed. When enough has been done, added destruction in an effort to get a dry surface is unwarranted. A slightly oozing surface may be dusted with an astringent powder but in many cases a protective dressing may be required, especially if the part is to be covered by clothing. When a dry scab is not readily procured, dressings should be changed and the wound cleaned during healing.

Galvanocautery. The galvanocautery is the most convenient cauterizing agent in dermatology. The cautery is applied for a fraction of a second when it assumes a bright heat, making repeated applications if necessary. The galvanocautery may be employed in cavernous nerves, which are too thick and deeply seated to permit removal by freezing with solid carbon dioxide.

Stellate and capillary nevus, telangiectases, small warts and moles, and lupus nodules may be cauterized under anesthesia. The galvanocautery is also ap-

This blister is left undisturbed until it spontaneously ruptures when the raw surface is covered with mild ointment.

Two or three applications of solid carbon dioxide may be necessary to obtain the desired result. The scar resulting from refrigeration is soft white and pliable. Better results are obtained by applying solid snow instead of an acetone-snow mixture.

Diathermy Diathermy may be medical or surgical. Surgical diathermy is employed in dermatology and includes electrodesiccation, electrocoagulation and cutting or charring.

1. **Electrodesiccation (fulguration)** is applicable in epithelial growths. It is, therefore, indicated for superficial lesions like warts, moles and keratoses. It consists in using the small high frequency spark through air and not in contact with the lesion.

2. **Electrocoagulation** is indicated in cavernous nevi, hemangiomas, and lymphangiomas which are too thick to obliterate by desiccation. It is also useful for the destruction of malignant growths.

3. Small projecting tumors and pedunculated new growths are excised by employing a loop electrode.

The electric cautery produces a hypertrophied scar. Electrodesiccation is followed by connective tissue hyperplasia.

Ether and oxygen should not be given as a general anesthetic while the diathermy machine is working.

Electrodesiccation is usually the method of choice for removing small skin lesions. This properly done gives the best cosmetic results, and should there be suspicion of potential malignancy it provides one of the surest prophylactics against recurrence.

The destruction is caused by heat, with no associated chemical changes such as occur with galvanism. The heat

should be limited to an amount sufficient to cause dehydration and produce no charring. This is a vital factor in avoiding undue fibrous tissue formation. Sections of desiccated tissue often show many remnants of cell and nuclear structure. Hard contracted scars and sometimes keloids follow severe burns, whereas soft and pliable scars or none at all result when there is just enough heat to produce devitalization.

The current used is of the Oudin type from a long wave high frequency machine. It is a monoterminal connection, the wire being attached to an appropriate holder with a sharp-pointed needle. A sharp point on the operating needle permits finer control of the amount of current than can be secured with the blunter tools often included in equipment of apparatus. This current is of higher voltage and lower amperage than that used for long wave diathermy or electrocoagulation and not all machines supply the most useful quality of spark.

Clark's original desiccation current was derived from a high speed static machine, with Leyden jars attached to the terminals and a solenoid connecting the outer coatings of the jars. This gave a comparatively cool spark, but developed enough heat for the destruction of small areas.

A spark-gap so constructed that a single gap may be used is preferred. To select the proper quality of spark start with the gap closed and the rheostat at its lowest operating adjustment. Open the gap slightly and test the spark by applying it to a metal object held in the hand. The heat is not felt in the hand. Then try it on the tip of the thumbnail. A brief touch causes only a slight sense of heat. A good way to determine if the spark has the proper quality is to apply

especially when employed after the age of eighteen years. Certain infectious diseases involving the hair follicle (*tinea capitis* and *sycosis vulgaris*) necessitate a temporary depilation which is accomplished by roentgen-ray irradiation.

DISADVANTAGES OF ROENTGEN-RAY THERAPY. Roentgen-ray therapy leads to pigmentation of the skin. The degree of pigmentation tends to vary in individuals. Overexposure to the roentgen rays is the only important drawback to irradiation. Overdosage may be attained by a single dose or by the cumulative action of small repeated doses.

Radio sensitiveness of the skin varies with sex, age, complexion, and the amount of blood present in the area undergoing treatment. Anemic surfaces are more resistant to irradiation. True idiosyncrasy to roentgen rays does not occur. The normal tissues particularly sensitive to irradiation are the testicles, ovaries, and lymphatics. All acutely inflamed and hyperemic lesions like acute eczema, acute psoriasis, leukemic, and pseudo-leukemic tissue are sensitive to irradiation.

The organs relatively sensitive to roentgen rays are the mucous membrane and the skin of infants and children, which is about 50 per cent more sensitive than that of adults. Connective tissue, cartilage, and bone are less sensitive to irradiation by roentgen rays.

Temporary sensitiveness may be due to local and systemic causes. The systemic causes include metabolic disturbances (gout and diabetes), blood diseases (chlorosis and eosinophilia), injections of tuberculin, vaccines, and general body irradiation by the quartz lamp. The local causes include irritation from chemical and quartz light and areas of scar tissue.

The reaction of the skin to roentgen ray irradiation does not follow the ex-

posure immediately but tends to occur after a definite latent period varying from eight to fourteen days. This latent period is relatively long after small doses and short following heavy doses. The action of a given measure of roentgen rays is greater when applied at a single sitting. The action on the tissue is less when the dose is divided into small individual doses or scattered over a wide area and given at intervals; however small doses given at short intervals before the tissue has had time to recover lead to a cumulative action of irradiation.

Irritating and local applications containing the metals are not applied to the body while the roentgen rays are in use. The drugs that can however be used with safety while irradiating with roentgen rays are vaseline, lanolin, alcohol, boric acid, zinc oxide, calamine and 1 per cent menthol.

Roentgen-ray irradiation in therapeutic doses tends to produce definite degenerative changes in the cutaneous tissue. The epidermis is the first to exhibit appreciable changes. Localized pigmentation and atonions are seen in patients treated over long periods. An erythema or a superficial dermatitis develops when the dose is pushed beyond a certain limit. The unfavorable sequelae vary from a slight erythema to an actual skin necrosis.

THERAPEUTIC DOSAGE. Unfiltered Doses. Andrews recommends irradiation by unfiltered doses of 100 kv unfiltered rays with an anode-skin distance of 8 inches, 2 milliamperes of current, and a spark gap of 11 inches for treating superficial lesions. An exposure of three minutes will produce a mild erythema. This irradiation is especially desirable in extensive lesions because it avoids the hazards of deep penetration. Extensive ex-

plicable in granuloma pyogenicum and for controlling hemorrhage in skin surgery

Galvanic hand and foot baths are useful in chilblains

Electrolysis is useful for removing superfluous hair, colloid milium, small moles, spider nevi, telangiectases, and xanthelasma. It is the best therapy for hypertrichosis. The negative pole is employed. From 1 to 2 ma. current is transmitted for fifteen seconds for the permanent destruction of hair papillae.

Ionization employing metallic radicals of zinc and copper salts is of value in lupus vulgaris, syccosis and the suppurative skin diseases.

Ultraviolet Light Therapy Ultraviolet rays are obtained from the sun, the carbon arc and the mercury vapor lamp. The direct application of ultraviolet light to cutaneous lesions is indicated in a few disorders. One of these is cutaneous tuberculosis, generally known as lupus vulgaris, and rosacea like tuberculi. Ultraviolet light is also invaluable in acne indurata. Direct irradiation by ultraviolet light increases local blood supply and is therefore of estimable value in alopecia and erysipelas. Ultraviolet light irradiation is helpful for its local and general effects in psoriasis and pityriasis rosea.

Pruritus is often improved by ultraviolet light therapy. It is especially helpful in local pruritus and in chronic eczema of the anus and scrotum.

Quartz light therapy is contraindicated in hydroa vacciniforme, xeroderma pigmentosum, pellagra, lupus erythematosus, in all individuals sensitive to light, and in cases of albinism and disordered hematoporphyrin metabolism.

The best technic for local irradiation is to place the lamp in a parallel position to the surface undergoing treat-

ment. The light is first applied at a distance of 60-80 cm. for a period of five minutes. The distance is reduced to 30 cm. and the exposure increased to fifteen minutes in the second treatment.

The water-cooled mercury vapor lamp is a small lamp possessing a quartz window. The heat produced by this lamp is absorbed by the circulating water enclosed in a jacket surrounding the burner. The water-cooled mercury vapor lamp is invaluable in tuberculous verrucous cuts and port wine angoma.

Large doses of ultraviolet light produce a marked dermatitis characterized by redness, swelling, vesiculation and pain. These symptoms are seldom serious and subside within a few days. A soothing liniment of caron oil or a zinc oxide paste with olive oil is all that is necessary to alleviate this dermatitis.

Röntgen Ray Therapy The physician should be acquainted with the applications of roentgen-ray therapy even though he is not in a position to employ it.

Röntgen-ray therapy is useful in treating dermatoses of pruritic nature such as pruritus ani, lichen planus, neurodermatitis, chronic eczema, and fungus infection because this form of irradiation tends to stimulate recovery. The x-rays are of value in the healing of ulcers and verrucous tuberculosis.

Röntgen ray irradiation increases the antibacterial action of the tissue. It is indicated in erysipelas, syccosis vulgaris, carbuncle, furunculosis, blastomycosis, warts, corns, calluses, and keloids.

The irradiation of malignant lesions by the roentgen rays tends to diminish the pain, lessen the discharge, and retard further progress of the disease. Sebaceous and sweat-gland secretions are diminished by roentgen ray irradiation. Acne vulgaris is improved by x-rays,

of the hard rays pass to the tissue. The extent of shielding the soft rays depends upon the quality and thickness of filter. The average thickness of filter used in dermatologic therapy ranges from 0.5 to 4 mm. of aluminum.

Soft rays are readily absorbed by the tissue, while the hard rays are feebly absorbed. A roentgen ray beam consisting of soft and hard rays loses most of its soft rays in the uppermost layer of the skin, where it exerts a more destructive action than an equal quantity of homogeneous hard rays.

The roentgen beam may be made harder and more homogeneous by suitable filtration so that less absorption takes place in the superficial tissue and irradiation is rendered of more equal intensity throughout the deeper tissues.

Filtered irradiation is especially useful in deep-seated lesions like subcutaneous nodules, malignant tumors, and diseases in which thick pathological tissue is present. There is little difference in the effect of filtered and unfiltered irradiation in the case of superficial dermatoses.

Indirect Roentgen Ray Therapy. Irradiation over the spinal ganglia has given relief in itchy cutaneous diseases and has occasionally relieved the pain of herpes zoster. Lichen planus has been treated by irradiating the sympathetic nervous system. The pruritus in Hodgkin's disease and in lymphatic leukemia is usually influenced by applying roentgen ray therapy to the affected lymph nodes. The technique for indirect roentgen-ray therapy consists of four doses of filtered 125 r ($\frac{1}{4}$ erythema dose) given daily or at weekly intervals.

Grenz Ray Therapy. Super-soft roentgen rays are known as grenz rays. Grenz rays are generated in a special tube at low voltage and of wave lengths of from 1 to 3 angstroms. They are em-

played for treating superficial dermatoses in which it is desirable to avoid the effects of the rays below the papillary layer of the dermis. This is especially desirable in treating diseases of the scrotum, scalp, and eyebrows because it is difficult to do harm with such rays, even with long heavy exposures. Skin previously irradiated with the ordinary roentgen rays is very sensitive to fractional doses of super-soft roentgen rays. The voltage should be 8 kv in grenz ray therapy. An exposure of 8 ma. for a period of four to six minutes with this voltage suffices to produce an erythema at a distance of 6 cm. from the center of the anode.

The therapeutic results from grenz rays are usually very good. Epilation cannot occur. Atrophy and telangiectasia are rare even after repeated large doses.

Radium. Various substances like thorium λ , radium proper and radium emanation are employed in therapy for their radioactive effect. The rays radiating from these substances are different from those of the roentgen rays in that a greater variety of rays are available for their therapeutic effect. The rays from thorium λ are chiefly alpha rays which are very soft and of slight penetrating power. Radium gives off both emanation and rays. The rays of radium are three: beta, alpha, and gamma rays. The alpha rays are the least penetrating and may be readily shielded by an aluminum screen of 0.01 mm. thickness. The greatest distance they can penetrate tissue is about 0.1 mm. The beta rays are of streaming electrons and are shielded by a lead screen of 10 cm. thickness. They are much more penetrating than alpha rays and can travel through tissue for a distance of about 10 mm., or 2 mm. of brass, monel metal, or steel. The

taneous areas are irradiated by a larger anode-skin distance ranging up to 10 inches or by overlapping fields.

Fractional Irradiation The maximum therapeutic effect of fractional doses is attained by applying repeatedly small doses of roentgen rays at short intervals over a considerable period of time. The intervals between treatments run over three days, one week, or two weeks. These short intervals do not allow the tissues to have time to recover from the effect of irradiation of one dose before being subjected to the following dose. Fractional doses of irradiation by the roentgen rays become intensive when repeated at intervals frequent enough to lead to cumulative effects. Treatments are not given at such long intervals that the therapeutic effect is lost or so close together that the accumulative effect produced damages the tissues. The skin is carefully observed for evidences of irritability when irradiation is continued over a period of more than a few weeks. The symptoms of irritability from irradiation consist of (1) a rapid hyperemia following stroking the body or after applying warmth to the skin (2) the onset of dryness due to the inhibition of sweat and sebum and (3) the appearance of epitheloma.

Simple acute and chronic inflammation of the skin involving the epidermis alone or the subpapillary zone responds to fractional unfiltered irradiation ranging from 50 to 100 r ($\frac{1}{2}$ to $\frac{3}{4}$ erythema dose) repeated every three to seven days.

Fractional weekly doses of 100 r ($\frac{3}{4}$ erythema dose) are applied in most cases of simple inflammation of the skin of adults. There is the danger of a systemic reaction in widespread diseases from treating more than circumscribed amounts of the involved areas with this

dose at one sitting. The total dosage is influenced by many factors in each case especially the size and location of areas to be treated and should never exceed 1600 r (four erythema doses).

Subfractional Irradiation Subfractional irradiation consists of smaller exposures of 50 r or $\frac{1}{2}$ erythema dose given once a week. It is applied in children for treating simple inflammatory dermatoses and is also employed in very acute inflammatory conditions of adults. This dose of 50 r may be applied on the scalp and eyebrows each week and repeated for a maximum of four weeks without the danger of epilation. An interval of at least six weeks should elapse before irradiation is again applied if epilation is to be avoided.

Intensive Irradiation Intensive or massive irradiation consists of applying one or more erythema doses. The maximum effect of irradiation is attained by one dose thus making repeated exposures unnecessary for some time. Intensive irradiation is only indicated in grave dermatoses, usually of the malignant type. When the lesions under treatment are small superficial and indolent relatively large amounts of unfiltered radiation may be given with proper shielding. The smallness of the exposed areas greatly reduces the actual dose. Unfiltered irradiation may be as high as 1200 r (three erythema doses) without danger of more than a second degree reaction. Successive treatments should not be given when massive doses are indicated until two or three weeks after the disappearance of erythema.

Filtered Irradiation A beam of rays of a roentgen tube consists of a mixture of soft and hard rays. A metal filter of aluminum placed between tube and patient's skin tends to hold back practically all the soft rays, thus making the bulk

roentgens (the unit originally adopted for expressing roentgen dosage)

When radium is used on the eyelids, the eyeball should be protected with a special convex-shaped brass or nickel-plated shield applied under the lid after cocainizing the conjunctival sac.

Radium is best avoided for lesions such as angiomata over the ovaries and testicles, on the scalp, and the epiphyses, especially of the fingers and toes.

Flat glazed applicators measuring 2 sq cm. and containing 10 mg. of radium are in common use by dermatologists. This standard 10 mg. of unscreened half strength radium plaque in direct contact with the skin will produce an erythema in about ten minutes. It will also lead to an erythema and temporary epilation in about twenty minutes. An intense erythema with desquamation and exudation is readily produced by this standard dose. Destructive reaction may develop with this standard dose of 10 mg. of unscreened half strength radium plaque in ninety minutes. The dose for children is reduced to approximately 50 per cent.

The erythema appears in a few days following treatment and continues as a mild reaction of the theme for a period of two weeks. Severe reactions last from six to ten weeks.

Applications of radon are employed in

the treatment of epithelioma instead of the flat glazed plaque just described

The chief practical advantage in radium therapy is that it can be used for lesions inaccessible to roentgen-ray therapy. It is especially indicated in treating such angiomata as *nevus vasculosus* and cavernous nevi. Roentgen keratosis, superficial carcinoma of the skin, Paget's disease, small tumors, keloids, warts, painful scars, and callouses are treated also by radium.

It is of much value in bringing on shrinkage and fibrosis in tuberculous adenitis. The removal of pyogenic granulomas with radium is usually successful, and if exposure is timed properly involution occurs in three to four weeks. Lupus vulgaris, when all other therapy has failed, is sometimes favorably influenced by radium. Moles, especially of the pigmented variety should not be irradiated.

Mesothorium is a radioactive substance separated as a by-product in the thorium industry. It owes 25 per cent of its activity to its radium content. Thorium X bears the same relation to mesothorium that radon bears to radium. It can be incorporated in ointments and lotions for treating telangiectases, eczema, psoriasis, etc. For additional discussion of x rays and radium, see chapter on cancer.

gamma rays are hard penetrating electromagnetic rays able to pass through a lead screen of 10 cm thickness. *Gamma rays* are of shorter wave length than most roentgen rays, ultraviolet light, visible radiations, and radio waves. They are generally emitted by the same atoms that emit beta rays. They are much more penetrating than either alpha or beta rays. A $\frac{1}{4}$ -inch lead filter reduces their intensity about 50 per cent and some rays can be detected through 6 inches or more of lead.

The term "radon" was suggested in 1923 by the International Committee on Chemical Elements to identify what is generally known as "radium emanation." One gram of radium produces 166 mc of radon per day.

Aluminum and lead screens are employed in radium therapy for shielding the undesirable radium rays because the beta and gamma rays are the only rays used. From 10 to 20 mg of radium is usually sufficient for treating ordinary skin lesions. This amount may be administered in a single application or may be divided into broken doses so that they may be applied each day for a few minutes or hours.

Radium as such does not have any therapeutic value. The tissue reactions which follow radium treatment are the result of absorption of energy by the tissue from the radiations emitted by radium and its disintegration products. The primary biologic effect is one of cell injury.

Radium is commercially available only in the form of a salt, generally the sulfate, bromide or chloride.

In ordinary radium treatment only the beta and gamma radiations are used.

The best method of applying radium and the most suitable dose in each case require training and judgment.

There are three general types of application: external, intracavitary and interstitial.

In external irradiation the radium applicator is either in contact with or at some distance from, the skin.

Plaque applicators usually contain from 5 to 25 mg of radium embedded in the surface of a glazed material having an area of from 1 to 8 sq cm. The filter in plaques is very small, seldom more than 0.1 mm of monel metal; hence most of the beta rays may be used. Plaques are generally placed in contact with the lesion to be treated and are used for surface lesions when irradiation is to be confined to a shallow depth.

Tubes containing radium generally have walls thick enough to absorb all the beta rays, so that only gamma rays may be used.

The dose of radiation delivered to the tissue depends on the amount of radium used, the length of time of application, the arrangement of the applicators, the filtration and the distance between the source and the tissue. The "biologic effect" produced by irradiation depends on the dose and many other physical and biologic factors, including the rate at which the energy is absorbed, the volume of tissue irradiated, the dosage distribution throughout that volume, the kind of tissue, the phase of the life cycle of the cells, the blood supply and many other factors.

The dose for radium salts is generally expressed in milligram hours (millicurie-hours for radon). To describe a treatment completely the amount of radium or radon, the time of application, the filtration, the distance from the lesion, and the type of applicator should be given. Present methods of expressing the dose are slowly being superseded by a simpler method in which the dose is stated in

mucocutaneous junctions are involved in about 50 per cent of cases. Hair loss, both of scalp and eyebrows, is not infrequent. In the malignant (adult) type, the eruption may precede definite signs and symptom of malignancy by many years, but rarely is the malignant neoplasm identified before appearance of the eruption. The plaques start as large hyperpigmented macules. The skin of the



Fig. 1 Acanthosis Nigricans, Juvenile type. Condition first noted 1 year of age. Patient is twenty five years of age at present time. Clear central area is site of biopsy. Note the papillary and pigimentary characteristics of this dermatosis.

involved region slowly becomes elevated, giving the appearance of exaggerated skin lines. The color of the involved region is a brownish gray. Involved mucosal regions are characterized by roughness with the formation of papillary or filiform excrescences. Itching is rarely present. Lesions of the two types show no gross or microscopic points of differentiation and they occur with equal frequency

Diagnosis Acanthosis nigricans should not be confused with Darier's disease in which the pigmentation is not nearly so marked. The scalp is usually affected with pseudopili in Darier's disease. The characteristic papillary hypertrophy of acanthosis nigricans distinguishes it from Addison's disease. The bronze pigmentation of Addison's disease is not marked over the face and neck. Verrucous lesions are absent in Addison's disease.

Prognosis The juvenile type of acanthosis nigricans is usually benign and does not apparently affect the health of the child. Cutaneous symptoms become less pronounced as maturity is reached.

The adult or malignant type is fatal.

Treatment The treatment is purely symptomatic in acanthosis nigricans of adults in which malignancy coexists. Muller reports a patient with adult acanthosis nigricans in whom the cutaneous symptoms disappeared after the removal of a growth.

Intense quartz lamp therapy is occasionally serviceable in the juvenile or benign type.

Cortical extract with sodium chloride has been recommended and is worthy of a trial. Ascorbic acid lessens the pigmentation. Potassium should be deleted from the diet and sodium chloride increased.

Röntgen therapy produces a flattening of the papillae and improves the appearance.

Frequentunctions of *Unna's* *peppermint* softens the skin and is, therefore, of service.

Phenol	10
Peppermint	50
Boric acid	10
Petrolatum	q.s. 100.0
Sig. Use 1/2 teaspoon followed by hot ap-	
plications	

ACANTHOSIS NIGRICANS

SYNONYM: *Keratosis nigricans*.

Acanthosis nigricans, or *keratosis nigricans*, is a rare skin disease characterized by the formation of soft velvety or verrucous plaques (papillary hypertrophy) and by hyperpigmentation.

Varieties The disease is divided into two types, the adult or malignant type associated with malignant tumors of internal organs, and the juvenile type in which malignancy does not occur.

Etiology Adult *acanthosis nigricans* is usually associated with malignancy. Mook and Brews found the undoubted presence of carcinoma in forty-three adults with *acanthosis nigricans*. They also reported twenty-two additional cases in whom new growths were probably present. The following distribution of malignancy was evident in the forty-three positive patients: namely, twenty-eight cases of carcinoma of the stomach, four cases of carcinoma of the uterus, two cases of carcinoma of the rectum, three cases of carcinoma of the breast, one case of carcinoma of the gallbladder, one case of carcinoma of the lung, one case of sarcoma of the colon, one case of chorioepithelioma, one case of lymphosarcoma, and one case of melanoma.

Schwartz and Miller champion the mechanico-nervous factor suggested by Darier who asserted "that intra-abdominal pressure from primary or metastatic neoplasm implicating the nervous system and causing an interference with its normal function plays an important part in the causation of the integumentary changes peculiar to the adult type of *acanthosis nigricans*."

Artom ascribes the etiology to developmental abnormalities and believes that

the coexistence of carcinoma depends upon the same constitutional factors.

Many clinicians are of the opinion that *acanthosis nigricans* is due to suprarenal disease. No cases have as yet been reported with Addison's disease in which *acanthosis nigricans* was present. The relationship if any of adrenal disease and *acanthosis nigricans* has not been established.

Kutter believes that the disease is idiopathic and that it occurs in young persons with familial obesity or diabetes.

The juvenile type of *acanthosis nigricans* reported by Knowles, Sidlick, and Judy presents certain clinical findings which indicate tuberculosis as the causative factor.

Pardo Castello and Mestre reported a twenty-three-year-old male with juvenile *acanthosis nigricans* in whom the disease was present since the age of three years. Clinical findings in this patient led them to ascribe the etiology to sympathetic and endocrine disturbances.

Pathology The histopathology of *acanthosis nigricans* reveals marked acanthosis and hyperkeratosis with elongation of the interpapillary projections and hyperpigmentation of the basal cell layer of the epidermis. There is little or no inflammation.

Symptoms The lesions are of symmetrical distribution. The regions affected in their frequency are the axillae, neck, genitalia, groins, arms, umbilical area, the flexor surfaces of elbows and knees, and the breasts. The palms and soles may show the presence of hyperkeratosis and the nails may be striated and brittle. The mucous membranes and

mucocutaneous junctions are involved in about 50 per cent of cases. Hair loss, both of scalp and eyebrows, is not infrequent. In the malignant (adult) type, the eruption may precede definite signs and symptoms of malignancy by many years, but rarely is the malignant neoplasm identified before appearance of the eruption. The plaques start as large hyperpigmented macules. The skin of the



Fig. 1 Acanthosis Nigricans, Juvenile type. Condition first noted at age of nine. Patient is twenty-five years of age at present time. Clear central area is site of biopsy. Note the papillary and pigmented characteristics of this dermatosis.

involved region slowly becomes elevated, giving the appearance of exaggerated skin lines. The color of the involved region is a brownish gray. Involved mucosal regions are characterized by roughness with the formation of papillary or filiform excrescences. Itching is rarely present. Lesions of the two types show no gross or microscopic points of differentiation and they occur with equal frequency

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Röntgen therapy produces a flattening of the papillae and improves the appearance.

Frequent injections of *Urea* or *peppermint* softens the skin and is, therefore, of service.

Phenol	1.0
Epain	1.0
Boric acid	1.0
Petrolatum	1.0
Use	q. s. 100.0
Application	with massage followed by hot ap-

ACNE

Acne Keloid

SYNONYMS: *Dermatitis papillaris capillitii sycosis nuchae folliculitis cheloidalis capillitii sycosis framboesiformis acne sclerolentans nuchae folliculitis keloidalis.*

Acne keloid is a chronic inflammatory disease characterized by the presence of papules, pustules, and hypertrophic scars (keloids) on the nape and adjacent parts of the scalp.

Incidence The disease occurs in adult life, most frequently in males, and particularly in Negroes.

Etiology The disease is due to the *Staphylococcus pyogenes aureus* and *albus*. Sabouraud regards the acne bacillus as the causative agent. Adamson believes that trauma is necessary for its production. Barber states that it occurs only in males who have seborrhea. A history of furunculosis is often obtainable.

Pathology The disease begins in the hair follicle and is only an advanced stage of sycosis vulgaris that takes on certain characteristics due to its location in tissues with different anatomical structure.

In the beginning of acne keloid the histology is characterized by endo- and perifolliculitis. As the inflammatory and cellular infiltrate increases the follicle and the surrounding tissue are completely destroyed and an abscess results.

The elastic and collagenous tissue within the infiltrated area is gradually destroyed and is replaced by scar tissue. Instead of terminating in necrosis, the folliculitis and perifolliculitis heal by means of a sclerosis of the connective tissue. The sebaceous glands are usually either completely destroyed or are atrophied.

As the sclerotic fibrous tissue limits

and replaces the abscesses, healing on the surface begins. The lesion is in some places covered by a narrow layer of new formed epithelium while in others there is hypertrophy of the rete and papillae leading to papillomata. Hairs located superficially in the cutis are completely destroyed at the same time as the follicle and sebaceous glands but the hairs having a deeper origin are frequently found to be spared.

Symptoms The disease begins with the formation of a few or several rounded or acuminate pin sized nodules at the hair line, or the nape of the neck. The nodules are reddish in color and firm in consistency. They occasionally coalesce and form keloidal plaques. They range from a pinhead to the size of a bean. When punctured they bleed freely. Pustules occur between the lesions and the entire group often becomes undermined and boggy with circumscribed cutaneous abscesses. These abscesses heal and leave scars which often become keloidal. Tufts of twisted, deformed and broken hair often project through the nodules. The disease is usually confined to the nape of the neck but its various manifestations may also involve the occiput, the chin and upper lip.

Differential Diagnosis This disease may be mistaken for ordinary keloids. Keloid is a noninflammatory fibroma histologically. In acne keloid one always finds islands of chronic inflammatory infiltrate especially plasma cells and elastic fibers. The location and keloidal tendency of acne keloid are so characteristic that other diseases are excluded without difficulty.

Prognosis There is no tendency to spontaneous cure. The process usually remains stationary after a variable time.

Treatment Irradiation by radium and x rays and applications of solid carbon dioxide offer the best methods of treatment. Radiation may be preceded with benefit, in some cases, by crisscross, superficial, fine incisions. Equal parts of *emplastrum plumbi* and *petrolatum* with 5 per cent tannic acid or 10 per cent salicylic acid are very helpful in mild cases of acne keloid.

Two applications each day of a 2 per cent solution of menthol in cottonseed oil are helpful in the keloidal stage of the disease.

Thiosmamin is a resolvent of connective tissue and is indicated in the treatment of keloids. It is given in doses of 100 mg every three days, using a 10 per cent solution in 80 per cent glycerin-water vehicle. It may also be applied with Iat-



Fig. 2 Left Acne Keloid. Mild form. Note the follicular character of the discrete lesions. (Courtesy of Dr. Jacques P. Carabacera.) Right Acne Keloid. Showing closely placed, practically confluent papules and pustules with fibrous tissue hypertrophy.

Daily applications of half strength tincture of iodine followed by a full strength solution of the tincture are used until the skin is dry and shows a tendency to flaking. This is followed by an ointment containing 3 per cent of each of sulfur and resorcin. Quinolone ointment may also be employed in the following proportions.

Chloroxydine quinoline	0.5
Benzoyl peroxide	10.0
Emulphor	1.0
Oleum Olus	1.0
Glycerin	q. s. 100

ac's paste as a vehicle thiosmamin 10 per cent salicylic acid, 2 per cent.

Acne Rosacea

SYNONYMS Rosacea, acne erythematosa, grana rosea, brassy face, ruddy complexion, lupus facialis, lupus erythematosus.

Acne rosacea is a chronic erythema involving the nose, middle portion of the face adjacent areas of the cheeks, forehead, and chin. It is characterized by the appearance of pustules and pap-

ules upon an oily erythematous skin

Incidence Acne rosacea occurs in middle life in individuals with seborrhea and neurovascular instability. It is more common in females.

Etiology The etiology of acne rosacea is closely allied to that of acne vul-

garis but differs in the age at which it occurs. Acne rosacea is seldom seen before the thirtieth year. The occurrence of acne rosacea in women about the menopause is unquestionably the result of the vasomotor instability accompanying this period of life. Indigestion due to improper food, improperly prepared

food or excessive indulgence in alcoholic drinks, tea and coffee are important etiological factors. Intranasal pressure or disease leading to vascular and lymphatic obstruction, syphilis, exposure to stormy weather, rapid changes of external temperature, great heat, endocrine

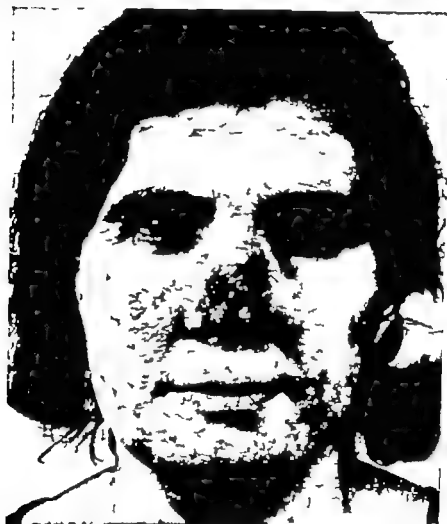


Fig. 3. Acne Rosacea. Showing rosacea associated with pustular acne and excessively large pores.

dysfunction and vasomotor neuroses are considered among the etiological factors. Hot foods and drinks, highly spiced dishes and beverages, iodides, bromides, and foci of infection are also causative factors of this condition. Achlorhydria and hypochlorhydria are frequently associated with acne rosacea. Ayres and



Fig. 4 Basore. With heretike. (Courtesy of Dr. Carroll S. Wright.)

Anderson have shown that the *Demodex folliculorum* is present in many cases of rosacea and they believe it has some etiological significance. The history in many instances, shows that flushing or flushing, on even the slightest emotional stimulation, has been present since childhood. It is possible that this habit, continued for years after most have learned to control it, has much to do with the development of rosacea. It is as if the repeated dilatation of the facial vessels has led to a degree of atony or persistent dilatation. This state is an erythema, but also enables the aforementioned etiological factors to become operative.

Pathology The first stage of acne rosacea is accompanied by a persistent hyperemia in consequence of which the capillaries become permanently enlarged. This leads to increased vascularity of the skin and ends in hypertrophy of the sebaceous glands and surrounding connective tissue. The fibrous hyperplasia impedes the circulation and in turn tends to block lymphatic drainage. The nose assumes a deep red or purplish color. Acneform lesions are practically identical with those of acne vulgaris excepting that the wall of the acne pustule is thinner. Lymphocytes and polymorphous clear leukocytes are more numerous than in acne vulgaris.

Symptoms Acne rosacea is characterized by erythema of the nose, cheeks, forehead, and chin. It is accompanied by seborrhea, telangiectasis, acne, and occasionally hypertrophy of the nose; the latter is known as rhinophyma. Acne rosacea usually begins on the tip of the nose with a chronic redness and seborrhea. The skin is greasy and shiny and telangiectasis is seen in the subepidermis. The process spreads to the cheeks, forehead, and chin, and acne-like pustules usually complicate the picture.

In the final stage, the skin of the nose becomes thick, coarse, and deeply congested and forms nodular masses. The condition is referred to as rhinophyma.

The ocular manifestations include keratitis, conjunctivitis, blepharitis, and occasionally iritis. Iritis may occur without corneal involvement.



FIG. 5. Rhinophyma.
(Courtesy of Dr. C. C. Thomas.)

Differential Diagnosis: Acne rosacea is not to be confused with acne vulgaris and lupus erythematosus.

The distribution of lesions of acne vulgaris is diagnostic. They occur before the age of twenty years and are accompanied with comedones. Acne rosacea seldom occurs before this age and is free from blackheads.

The affected skin tends to become dry and atrophic in lupus erythematosus. The skin is oily and hypertrophic in acne rosacea. Pustules are common in acne

rosacea and rare in lupus erythematosus. The characteristic patch of lupus erythematosus is sharply defined and diffuse in acne rosacea. Scaling and atrophic scarring are usually present in lupus erythematosus but absent in acne rosacea.

The central predilection and confluence of acne rosacea, as well as the age of the patient, the course of the disease, the absence of oozing, itching and ulceration exclude papular dermatitis, lupus vulgaris, syphilid, tuberculids, lupus miliaris disseminatus faciei and dermatitis seborrheica.

Papular acneform lesions may develop in the course of rosacea, but there are no comedones. Such papules histologically may even suggest a tuberculous architecture. The condition clears rapidly, however, under the usual therapy for rosacea, but rosacea like tuberculid and lupus miliaris are apt to be protracted and recalcitrant. The differential diagnosis in all three conditions may depend on the characteristic histologic reactions, the tubercle-like structure without necrosis in Lewandowsky tuberculid and a similar structure in lupus miliaris but with necrosis. In these types of rosacea, the differentiation from the Lewandowsky rosacea like tuberculid and lupus miliaris disseminatus faciei may be clinically impossible. (The tuberculin test is apt to be negative in both of these conditions.)

Rosacea must also be distinguished from granuloma rubrum nasi which usually occurs in children and is characterized by a diffuse redness of the lower portion of the nose, especially tip and alae, and sometimes the chin. It is generally accompanied by pinpoint dark red papules. A localized hyperidrosis is constant. It is chronic and tends to disappear with age.

Complications. The most serious complication of acne rosacea is ulcerative keratitis. Occasionally the ocular complications occur without any cutaneous manifestations.

Prognosis. Acne rosacea is a chronic disease. The mild types are usually amenable to treatment. The hypertrophic form can be greatly improved and disfigurement reduced by surgical procedures.

Prophylaxis. Corrections of digestive disturbances, elimination of foci of infection, avoidance of alcoholic drinks, and protection against rapid changes of temperature are prophylactic measures and should be instituted before the advent of early middle life.

Treatment. The golden rule in treating acne rosacea is to ascertain and then remove the underlying cause. Dyspepsia is the most offending agent, although local sepsis in the nose and sinus can produce it. Dyspepsia usually improves with the avoidance of tea, coffee, alcohol, and indigestible articles of food.

The reflex which dilates and finally paralyzes the blood vessels of the flush areas of the face must be controlled or abolished. Anything which causes flushing or blushing must be avoided. A conscious effort on the part of the patient to stop the emotional factor generally leads to its control in six to eight weeks. Among the flush stimulants are rapid eating, coffee, tea, alcohol, hot foods and drinks, condiments, rich foods, pastries, and gravies or exposure to heat in kitchens, and these must be avoided.

The ingestion of dilute hydrochloric acid in water with the meal, or the intake of such alkalis as sodium bicarbonate or magnesium oxide one to two hours after the meal are helpful to some patients. One-half teaspoonful (20) of dilute hydrochloric acid in 2 ounces

(90.0) of water after meals and repeated in twenty minutes, is highly beneficial in cases of hypochlorhydria.

Frederick R. Schmidt, in the Medical Clinics of North America (Jan., 1937) found the following prescription a valuable aid to digestion

Hydrochloric acid	100
Pepsin scales	100
Dist. lacte pepsin	q s. 1000
Sig. One teaspoonful in water after meals, taken through glass tube.	

Salted and highly seasoned articles of food, such as sausages, smoked meats, cheese, and white bread and butter should be eliminated. A diet consisting of fruit, vegetables, carbohydrates, with a minimum amount of fat and protein is helpful to this may be added 300 gm. of meat and two eggs per week in cases where additional calories may be necessary.

Enteric-coated pancreatic enzymic tablets are worthy of trial.

Endocrine therapy is indicated in some cases of acne rosacea. Two grains of desiccated thyroid gland given once daily and continued until signs of intolerance develop has proved helpful in the author's cases.

In ocular complications, riboflavin is promptly effectual.

The local treatment of acne rosacea is much the same as that of acne vulgaris. Soaps are usually irritating and should, therefore, be avoided. Freshly prepared lotio alba (zinc sulfate and potassium sulfate) is suitable for ordinary cases of chronic acne rosacea. Two applications, each day of the following ointment have proved very helpful in cases associated with pustules.

Pol. camphor	0.5
Ichthyol	10
Colloidal sulfur	40
Glycerol cream	300

Fac. Apply at bedtime

The face skin may be peeled with equal parts of resorcin and Unna's soft zinc paste. This keratolytic paste is applied for three successive days, followed by compresses of hot boric acid lotion. The patient is confined during this treatment which accomplishes more in a week than milder treatment would in a month. Viciously dilated cutaneous capillaries can be destroyed by electrolysis or by galvano-cautery. With electrolysis the needle is attached to the negative pole, inserted into the vessel and a current of 1 to 1½ ma. is allowed to flow for several minutes or until the vessel turns white. To prevent bleeding, the needle is removed while the current is still flowing. This treatment may have to be repeated. The area may also be criss-crossed superficially with a fine scalpel, and scarring does not occur. Calamine lotion with 2 per cent ichthylol is useful in treating the erythema. Rhinophyma is treated surgically.

Heliotherapy, ultraviolet irradiation, and the x rays are only occasionally beneficial. Moderate refrigeration by means of solid carbon dioxide and acetone slush is highly recommended by some authors.

If acne rosacea is associated with dandruff or a seborrheic dermatitis, treatment for them is necessary before a cure or improvement may be expected.

Acne Varioliformis

SYNONYMS *Acne necrotica*, *acne atrophica*, *acne with depressed cicatrices* (Heister) *acne radens*.

This is a chronic and persistently recurrent affection, characterized by discrete or grouped pinkish red papules, papulopustules, papulocrusted elements, and varioliform cicatrices. The papules are the size of a pinhead or slightly larger.

Etiology Commonly observed in the adult and in the male more often than the female the lesions are in all probability due to bacteria acting on a specially predisposed skin—often *seborrheal*. According to Sabouraud the condition is



Fig. 6. *Acne Necrotica*. Extensive infection. Not numerous violiform scars on ten pla. cheek and nose.

due to the symbiotic action of the microbacillus of *seborrhea* and the *Staphylococcus aureus*. However Strumia has experimentally produced typical lesions in a patient by means of intradermic injections of staphylococci and streptococci obtained from lesions in a patient with extensive skin involvement.

Symptoms Itching and burning sensations at the sites of the lesions are frequent. Mild superficial and abortive types are not uncommon particularly on the scalp and in the *fastidious*. In these, persistent or recurrent attacks of itching and a few discrete often perifollicular lesions on the scalp are the only evidences of the disease which has been described under the term "*acne*

necrotica miliaris." The severe type is not common. The sites of predilection are scalp, temples, forehead (especially at hairline) and sides of the nose, and it may be limited to any one of these areas. In some the lesions are numerous and extensive and even the trunk—anteriorly and posteriorly—and extremities are involved. The characteristic lesion often pierced by a hair is a pinkish red inflammatory papule the center of which rapidly becomes yellowish flat



Fig. 7. *Acne Necrotica*. Extensive infection of scalp, forehead, nose and cheeks. Note the active crusted-covered, pinhead-sized lesions and the scars of previous lesions.

tens, and dries to a depressed brownish and very adherent crust. After a variable period—ten to fourteen days—the crust falls and exposes a pink ultimately

white, small depressed and varioliform scar. In patients who have had the condition for years, numerous such pinhead and smaller sized scars are present. The disease persists as a result of the development of new lesions.

Acne Necroticans Exulcerans Serpiginosa Nasi (Folliculitis Exulcerans) of Kaposi. This variety of acne necrotica is a rare affection which develops on the nose and ala nasi in the form of soft pink papules which rapidly enlarge and undergo supuration. The pus is discharged and neighboring lesions join to form deep serpiginous ulcers with soft borders. These heal in several months, leaving irregular cicatrices.

Diagnosis. The diagnosis is marked in cases is not difficult. The lesions in ordinary acne vulgaris do not itch or have varioliform scars; differentiation may be made from secondary syphilides by the location, central necrosis, the varioliform cicatrices, and other signs of a phylis.

Prognosis. Untreated, it persists for years. Relapses and recurrences are common. When properly treated, good results are quickly obtained.

Treatment. Local treatment early cures this affection. The treatment should be continued for six to eight weeks after the last lesion has disappeared. *Ointments* containing 5 per cent ichthammol, ammoniated mercury, sulfur or resorcin are efficacious. For the first ten days, the scalp and affected areas should be thoroughly washed with a tar or sulfur-containing soap and the following creams rubbed into the skin:

Salicylic acid	10
Prep. sulfur	10
Oil of rose	10
Greasy emulsion base (Vaseline)	300

Recurrences are prevented by frequent shampoos and the application

three times weekly of a lotion containing sulfur or bichloride of mercury 1:1000.

The following lotion is useful:

Sulfur	150
Glycerin	50
Camphor	20
Alcohol	250
Witch hazel	100
Bol. phenacrol (1:1000)	q. ad. 1500
Eva. Sibaka.	

Acne Vulgaris

SYNONYMS. *Acne simplex, acne discolorata.*

Acne vulgaris is a chronic inflammatory condition of the sebaceous glands characterized by comedones, papules, pustules, tubercles, and nodules, or a combination of all of these lesions. It appears chiefly on the face and infrequently on the shoulders, chest, and back. The disease is usually associated with oily seborrhea and pityriasis capitis (dandruff). The commonest lesions are comedones, papules, and pustules.

Varieties. For the convenience of description, various clinical types are named. The predominant type of lesions present, the accidental or coincident character of the inflammatory process and the resulting changes suggest the names for the several varieties as follows:

Acne Simplex. In this type the lesions consist of a few papules and pustules which are very superficial.

Acne Punctata. In this type the predominant lesions are comedones (black heads). An individual with this type of acne shows a considerable degree of seborrhea, the pores of the nose and of the surrounding skin are enlarged and contain a whitish mass of sebum. Little, if any inflammation is present. In children, comedones often occur in groups on the back and chest which are not associated with other lesions commonly

seen in acne. Comedones in children may be caused by the application of camphorated oil or liniment.

Acne Papulosa In this variety the inflammatory process is more pronounced. This type of acne is most common in males with coarse oily skin. A large number of comedones are usually present. The papules however predominate

tion. The lesions are deep seated and destructive and may be papular, pustular or both. Focal infections and systemic disorders are etiological factors of this type of acne.

Acne Conglobata This is a rare form of acne in which comedones, papules, and pustules are accompanied by large fluctuating plaques which are dusky blue



Fig. 8. *Acne Vulgaris*. Not comedones, papules, and pustules. A polymorphous eruption characteristic of this disease.

and are most frequently situated on the forehead, often on an inflamed base.

Acne Pustulosa This is a type of superficial acne which resembles a staphylococcus dermatitis. It occurs most frequently in young girls with a delicate skin. There are few if any comedones associated with this type of acne.

Acne Indurata Papular acne often progresses into an indurated type, forming inflammatory nodules which show no tendency toward spontaneous suppuration (blind boil). The skin is usually sallow, boggy and very oily. It results from a secondary staphylococcus infec-

tion. Cutaneous and subcutaneous abscesses and oil cysts are present and occasionally form discharging sinuses. They heal slowly and often leave keloidal or so-called "bridge" scars. Although this type of acne resembles scrofuloderma, it is the consensus that tuberculosis plays no part in its causation. The lesions are autoinoculable.

Acne Cacheticorum This is a variety of acne which is often seen in cachectic patients suffering from such chronic diseases as pulmonary tuberculosis, bronchiectasis, lung abscess, diabetes, anemia, etc. It is characterized by indolent, soft,

slightly infiltrated, pustular purplish lesions. Inflammatory reactions or scarring are usually absent. Comedones are absent or only few in number.

Acne Cystic. This type of acne is characterized by the presence of cysts varying in size from that of a lentil to

atrophic pits and scars. These lesions occur at sites which were formerly the seat of small papular lesions.

Acne Hypertrophicus. This type of acne usually follows acne indurata where slight connective newgrowth has developed. The scar is may be keloidal.



Fig. 9. *Acne Vulgaris.*

a hazelnut. This type is either associated with acne indurata or is accompanied with an oily seborrhea or impregnated comedones. The contents are jellylike, and consist of a granular serousanguinous fluid and inspissated sebum.

Acne Atrophicans. This type of acne is characterized by the presence of tiny

Acne Colloid. This is characterized by papular lesions occurring chiefly on the cheeks and forehead. The lesions, an incompletely involutioned pustular acne, are yellow transparent and colloid in appearance. If the lesion is punctured, a yellowish, jellylike substance exudes. This is followed by cure.

Acne Artificialis This is a term applied to the acne-like eruption developing from extrinsic causes. Workers in tar, chrysarobin, oils, coal tar distillates, lime, zinc, and flax often develop acne artificialis.



Fig. 10: Acne Conglobata. With complicating rosacea. (Courtesy of Dr. Carroll S. Wright.)

These several artificial eruptions simulating acne are described under *Dermatitis medicamentosa*.

Acne Corporis In exceptional instances, a large portion of the body may be covered with acne. The underwear may be a causative factor and should be kept scrupulously clean.

Acne Excorée (Excoriated Acne) This is a pruriginous dermatosis characterized by the development of very itchy papules on the face and neck of young girls and nervous women. It is allied to tic or nailbiting. Seborrhea and comedones are usually absent. Males are rarely affected.

Incidence Acne vulgaris affects the majority of adolescents to a greater or

lesser extent. It usually occurs between the ages of twelve and thirty; exceptionally it begins earlier and not infrequently it persists after the latter age. It rarely is seen for the first time after the age of thirty and is common to both sexes in all walks of life. It is more frequent in individuals of light complexion and in those leading a sedentary life.

There is a distinct tendency for severe acne vulgaris to appear in certain families. Familial association of acne vulgaris with early baldness in the male has been noted. This is of interest in relation to the theory of gonadal origin of both baldness and acne, and to the fact that at puberty the pilosebaceous apparatus of the male evidences a greater reaction



Fig. 11 Acne Conglobata. Not bridg scar of Lang on forehead.

to hormonal stimuli than does that of the female.

Etiology The etiology of acne is not definitely understood; it is thought to be partly bacterial (acne bacillus and staphylococcus) and partly constitu-

tional (endocrine disturbance, disorders of the gastrointestinal tract and pelvic organs, faulty diet, foci of infection, hygiene, etc.)

Chocolate, cocoa, and fermented cheeses are definitely harmful to patients with acne and also may be etiologic factors. The halogen drug group (iodine bromine chlorine) and tuberculin are also causative of acneform lesions. Potassium bromate present in the improver of white bread is reduced in baking to bromide and so white bread may produce some acne vulgaris. Iodized salt and foods rich in iodine are also regarded as acne-producing agents. Thyroid medication given for other conditions may acerate or even cause acneform lesions.

The affection is due to changes in the pilosebaceous glands and it is intimately connected with that of seborrhea. Bloch believed that early seborrhea is related to the physiologic function of the sexual glands and that variations in degree are due to a difference in sensitivity of the pilosebaceous glands to the sexual hormones. This relation is also manifested in women where definite aggravation of acne often occurs just before, during or immediately after menstruation. The relation is further emphasized by the fact that eunuchs have neither seborrhea nor acne; that a preexisting acne will disappear upon extirpation of the gonads and that acne after the climacterium is a rarity.

Lawrence considers the endocrine imbalance incident to adolescence the main etiologic factor in acne vulgaris. He believes this imbalance is in the anterior pituitary-gonadal mechanism and that the sex glands of acne patients are apparently functioning immaturely. The beneficial effects of aututrin "B" depend in this case upon the maturative influence of this hormone.

In practically all of the lesions, comedones as well as papulopustules, a microbacillus (acne bacillus of Sabouraud) is found. In addition to this organism, various kinds of staphylococci have been demonstrated, the most common of which is the *Staphylococcus pyogenes albus*.

Ramel has recently suggested the possibility that tuberculosis may be one of the etiologic factors in some of the varieties of acne.

Neurocirculatory instability (nervous breakdown, sexual tension) is often present in acne. Repeated blushing has a harmful influence in acne vulgaris and may be a prime factor in its persistence.

Pathology The pathology of acne lesions varies considerably with the stage of development. In the papular type of acne, we note a follicular and perifollicular inflammation. The follicular epithelium is edematous and swollen and is invaded by polymorphonuclear leukocytes. In the neighborhood of the follicle and the sebaceous glands, the blood vessels are greatly dilated and surrounded by an infiltration of lymphocytes, polymorphonuclear leukocytes, and occasionally plasma cells. At this stage of development, the pathology is that of a pseudofolliculitis. The papules may remain as such and gradually heal without pustulation. If pustules develop the epithelium is invaded by leukocytes, the cells are widely separated and the nucleus does not stain well and appears fragmented and the cell protoplasm is finally destroyed. We now have supuration and after evacuation of the abscess there remains a large space formerly occupied by the follicle and its appendages. Because of the pressure from the comedone and the retention of the increased sebaceous content of the follicle, the covering epithelium becomes

atrophic and finally breaks down and the follicle empties itself of comedone, pustular contents, and cell debris. Granulation tissue is then formed and later develops into a scar. If this acute inflammatory process is repeated in the same area an acne indurata results.

The changes in acne vulgaris are summed up by Lee McCarthy as an



Fig. 12. Acne Vulgaris. A deep pustule and cystic form with marked scarring. Entire back, nape of neck, and face in old. Courtesy of Dr. Jacques L. Guequiere.)

endo- and perifolliculitis of the lanugo hair follicle and its appendage the sebaceous gland the latter often being involved to a much greater extent. These changes manifest themselves in the following stages: (1) Formation of comedone (2) dilatation of follicle on account of increased sebaceous content and resulting thinning and atrophy of the epithelial wall (3) papule formation (perifollicular inflammatory cellular infiltration and beginning proliferation of connective tissue cells) (4) pustulation

and pressure atrophy of the epithelial roof of follicle, (5) expulsion of contents of follicular and perifollicular abscess; (6) granulation of cavity and repair by scar tissue.

Large pores, common in oily and acne-infected skins, may be due to mechanical blocking and subsequent dilatation at the oil duct orifices by hardened sebum. Kromayer states, however, that the pores themselves are not involved but are set deep in a skin in which the connective tissue, both beneath the epidermis and between the orifices, has become increased. The pores are best improved by peeling the skin with strong ultraviolet radiation.

Symptoms. Subjective symptoms except in some special forms of acne such as acne excoricee where itching is predominant are absent. There may however be slight itching of the face and when the inflammatory process is acute some tenderness and pain may be present. If the lesions occur on the forehead or directly over a bone this is usually the case.

Objective Symptoms. The skin is usually greasy and often coarse and muddy in appearance. The commonest site for the disease is the face and it is usually limited to this region. Occasionally lesions occur on the neck, shoulder and back and in rare instances the eruption may be limited to the back and anterior surface of the trunk.

Multiform lesions ranging from a comedone unattended by inflammation to superficial abscesses are observed. The eruption generally comprises comedones (blackheads) and small pale red bright, or dark red pinhead to pea sized pustules and papules in various stages of evolution and involution.

The lesions may be acute in character with a hyperemic inflammatory base

or they may be sluggish in appearance. In the course of several days or a week, the early lesions disappear without leaving more than a reddish stain which finally fades. During this time, however, new lesions appear which go through the same evolution and involution stages and the disease becomes chronic.

After the age of twenty-five, if the general health is good, there is a tendency for the disease to subside spontaneously. Usually no scarring results and the patient recovers without a trace of the previous eruption. This is, however, not always the case, as slight tissue destruction or atrophic changes produce permanent scars. If suppuration is extensive, the areas are likely to be deeply pitted, and often nodular thickenings are left in the skin.

Diagnosis. The diagnosis of acne vulgaris presents little difficulty. The lesions only occur on those parts of the body where sebaceous glands are present, and those regions of the skin where the sebaceous glands are larger as on the face and forehead, ears, chest, and back. All doubts as to diagnosis will be eliminated if the characteristic lesion (comedone) is present or if the history of its presence is obtained. The association of oily seborrhea, comedones, pustules, nodules, and deeply seated furunculoid infiltration is diagnostic. A history of chronicity is usually obtainable.

Differential Diagnosis. Sulzberger and W. H. differentiate the following acneform eruptions from acne vulgaris:

1. Acneform drug eruptions.
2. Acne eruptions resulting from external agents.
3. Acneform tuberculoderm (rosacea-like tubercloid lupus miliaris disseminatus faciei, papulonecrotic tuberculid).
4. Rosacea (with or without accompanying acne rosacea).

5. Acne of the menopause of pregnancy of endocrinologic diseases (adrenal and anterior pituitary tumors, etc.).

6. Acne resulting from administration of sex hormones, particularly male sex hormones.

7. Acneform eruptions due to hypovitaminosis A and C.

8. Acne varioliformis.

9. Neurotic excoriations.

10. Dermatitis nodularis necroticans.

11. Furunculosis.

12. Sycosis vulgaris.

Acneform Drug Eruptions. The most common acneform eruptions of internal origin are the ones produced by the ingestion and injection of iodides or bromides. Iodide acne may follow the administration of very small doses of the drug, while some persons tolerate enormously large doses without producing any cutaneous symptoms. In iodide acne comedones are absent and the lesions appear simultaneously and are of the same type. The lesions of iodide acne as well as those arising from bromide are usually bright red and are not limited to any one part of the body. Bromide acne, like iodide acne is produced by the sensitization of the pilosebaceous system through which these drugs are eliminated. The lesions following the use of bromide do not arise simultaneously and are therefore not of the same age as those of iodide acne. The color of bromide acne is brown and the lesions have a tendency to grouping. These groups of lesions often coalesce and may form a scurpious or a frambeiform plaque. The lesions of bromide eruption may appear long after cessation of the drug.

Acne Eruption Resulting from External Origin. The acneform eruptions produced by tar, mineral oil, chlorine, halo-

wax or chloronaphthalene, are characterized by their irregular distribution and occur more frequently on the forearms and genitalia where acne vulgaris does not occur. In chloracne, there is a history of working around chlorine or chlorinated hydrocarbons. Numerous



Fig 13 Iodide-Induced Acneform Eruption: "Iodide acne"

comedones, indurated papules, and actual pustules occur.

Acneform Tuberculoderma. The absence of comedones and of seborrhea and the occurrence of crops of lesions with a dry necrotic plug in the center suggest the diagnosis of tuberculid rather than acne vulgaris. The tuberculous structure of lupus miliaris disseminatus is so characteristic and so definite that a histological differentiation between them offers no difficulty.

Acne Rosacea. The most important disease with which acne vulgaris is confused is acne rosacea. Rosacea appears

later in life, comedones are absent and pustules and telangiectasis are frequent. Even if both acne vulgaris and rosacea are associated, the distinctly more erythematous nature of the latter supports the diagnosis of acne rosacea.

Acne Due to Gynecopathic or Endocrine Disturbances. The acne of the climacteric of pregnancy and of endocrine disease is characterized by paucity of lesions, absence of comedones and the uniform type of lesions present. Concomitant signs of menopause, pregnancy or endocrine disease are pathognomonic.

Acne Due to Sex Hormones. In the acne resulting from the use of sex hormones the signs as noted in the preceding paragraph are diagnostic.

Acne Due to Hypovitaminosis A or C. The acneform eruptions resulting from hypovitaminosis A or C are distinctive. In vitamin A deficiency the acneform lesions occur usually in children, boys at or before puberty, and are more likely to occur in the summer months. The skin is darker than normal and is of a dull slate color. Keratotic facial comedones occur on the face while spiny papules are present on the anterolateral aspects of the thighs and extensor surfaces of the forearm. Decrease or absence of sweating and sebaceous secretion is characteristic of hypovitaminosis A.

In vitamin C deficiency follicular hyperkeratotic papules occur frequently on the inner aspects of the thighs and upper arms. Purpura, spongy and bleeding gums are often associated. Perifollicular hemorrhage is often associated with the hyperkeratotic papules.

Acne Varioliformis (Acne Necrotica). This is characterized by lesions which are either pustular or nodular which later become a pustule and are located on the scalp, temporal region and the

forehead. Itching is a common symptom. The center of the lesion becomes necrotic and forms a crust which on falling off leaves a depressed varioliform scar. These symptoms differentiate it from acne vulgaris.

Neurotic Eruptions. In neurotic patients, these are characterized by a number of very itchy excoriated, self-traumatized papules on the face and neck and occasionally elsewhere. Comedones and seborrhea are invariably absent.

Dermatitis Nodularis Necroticans. This condition is characterized by a chronic recurrent symmetrical polymorphous eruption occurring chiefly on the buttocks and extremities. Necrosis and hemorrhage are prominent features although vascular eruptions predominate. It often ends fatally. The symptoms of acne vulgaris show no similarity to the disease and difficulty in differentiation should not occur.

Furunculosis. A furuncle or boil is an inflammatory nodule which becomes a pustule with central necrosis followed by suppuration and scarring. The course varies from one to three weeks and the disease has no sites for predilection. These features differentiate it from acne vulgaris in which the lesions are multifocal and are of a chronic character.

Sycosis Vulgaris. This condition is characterized by the appearance of whitish pustules occurring on an erythematous base. It is a follicular pustular dermatosis almost always limited to the bearded region and occasionally to other hairy parts of the body. Absence of comedones and multiform lesions differentiates it from acne vulgaris.

Complications. With the possible exception of erysipelas complications of acne vulgaris do not occur.

Prognosis. In the majority of patients acne disappears spontaneously as

adult age is reached. Appropriate treatment shortens the duration of the disease and lessens the amount of scarring. Walker states that the main factors in the cure of acne are "time, health, and perseverance."

Prophylaxis. The elimination of constipation and the correction of gastrointestinal disturbances that may be present prior to the age of thirteen is essential for preventing acne. Dandruff and the oily seborrhea usually associated with and preceding acne must be treated. Daily applications to the face of an aqueous solution of borax ($\frac{1}{4}$ teaspoonful of powdered borax to a pint of water) correct any oily seborrhea that is present and diminish the possibility of the appearance of acne. Dandruff can be eliminated by applying to the scalp a lotion containing 2.5 per cent salicylic acid in alcohol with or without the addition of glycerine or oil, depending on whether the scalp is dry or excessively oily.

Excessive intake of starches, fats, and chocolate should be avoided. A moderate amount of outdoor exercise is advised and exposure to sunlight is definitely a prophylactic measure.

The face should be washed twice daily with hot water and soap, the skin should then be dried with cold water and thoroughly dried. If the foregoing methods fail to prevent the appearance of acne, the activity of the oil glands may be reduced by the use of roentgen rays. I have, in some resistant cases, succeeded in achieving a good result by abolishing the reflex blushing habit as noted under "Acne Rosacea."

Treatment. Acne is a disease whose social and economic aspects are of great importance. An unsightly complexion is often the cause of unemployment and of an inferiority complex.

Since we are still ignorant of the exact cause of acne, it follows that both systemic and local treatment are essential in most cases.

Systemic Therapy. Systemic treatment must be determined by a careful study of the patient. Any gastrointestinal disturbance must be corrected. Constipation must be corrected. In cases of gastric hypoacidity dilute *hydrochloric acid* should be given and in hyperacidity *alkalis* are indicated. A high protein diet is recommended; an excess of carbohydrates should be avoided. The following foods are harmful and should be prohibited: chocolate in any form, nuts of all kinds, cheese, pork, white bread, potatoes, rice, refined cereal, iodized salt, oranges and tomatoes.

Many young patients are drinking too much milk, or cocoa, malt, or chocolate milk, and eating too many peanut butter sandwiches. Milk if taken at all should be taken between meals rather than with a meal, and peanut butter should be entirely avoided.

Daily exposure to the direct rays of the sun or the use of the ultraviolet lamp both on the entire body and locally is advantageous.

Foci of infection must be eliminated.

If acne is aggravated during the menstrual cycle, injections of *estrogenic hormone* are beneficial. Lawrence and Feigenbaum report the successful treatment of acne with *antuitrin S* (an anterior pituitary hormone). They advise an initial dose of 1 cc (16 minims) of antuitrin S (100 rat units) to determine the patient's tolerance. If no intolerance is evident, he gives 2 cc three times a week until three or four days prior to the next menstrual period. Anemic patients should be given treatment with *iron arsenic* or *liver*. Sulzberger and Wolf prefer to administer arsenic by

injection and advise the following prescription:

Sodium arsenat	2.0
Phenol	1.0
℥q dist	q.s. ad. 100.0

M Dispense in a sterile rubber dam covered Lottle.

Sig. One drop subcutaneously. Increase the dose by 1 drop daily until 1.20 drops are given. Then reduce dose 1 drop daily.

Fowler's solution is of distinct value in acne conglobata.

Sutton and Sutton advise giving 2 grains of desiccated whole gland *thyroid* with the evening meal. The dose of thyroid must be adequate and just below the dose which produces symptoms of intoxication. After a few weeks the need for this dosage may no longer be present and the dose may then be halved or omitted.

Vaccines bacteriophages, and *toxoids* are of little if any value. The occasional good results obtained by their use are no doubt due to nonspecific protein therapy.

The intramuscular or intradermal injection of *sterile milk agan* or the patient's own blood is sometimes helpful and should always be used in the indurated types of acne. *Irosterol* in doses of 20,000 to 100,000 units daily has been followed by improvement in many cases.

Vitamin B complex increases the hydrochloric acid contents of the stomach and corrects constipation and is therefore helpful in treating acne. *Riboflavin* and *niacin* are also remedial measures.

Intravenous injections of *normal saline solution* or the oral administration of enteric-coated tablets of *sodium chloride* (15 gm. every two or three hours) is valuable in treating pustular acne in which bromides or iodides are the etiological factor. Normal salt solution may also be employed with benefit if it is directly injected into the pustule and around the inflammatory base.

In acne conglobata and resistant cases of cystic and indurated acne, penicillin by injection should be tried, or sulfa guanidine over a prolonged period may be helpful. Surgical drainage of the abscess cavities is necessary irrespective of the chemical or biologic therapy used.

Intramuscular injections of *manganese butyrate* and the oral administration of the various salts of tin have been useful in pustular acne.

Local Therapy Local treatment of acne vulgaris varies with the type.

The use of cosmetics, facial creams, vanishing cream, heavily scented or colored face powders, contact with oil, grease, tar and the wearing of rough, hairy or woolen garments, should be prohibited. Free sweating should also be discouraged and erotic preoccupation should be avoided. Acne vulgaris often disappears after marriage and often reappears after a divorce.

Acne patients should wash the face with hot soapy water just before retiring and rinse it with water as hot as can be borne. It should then be massaged

with the thumb and index finger for the purpose of expressing the oil from the enlarged pores. After this procedure it is again washed with hot water containing 2 cc ($\frac{1}{2}$ teaspoonful) of borax to the pint (500 cc.) of water and finally rinsed with very cold water and then thoroughly dried.

Comedones Comedones may be removed by applying cold cream containing 3 per cent resorcinol after the lapse of one hour a towel wrung out of steaming water is laid on the face. On removal of this hot compress, the blackheads are easily removed by a gentle downward pressure of the fingers. The patient's face may also be exposed to a wide-mouthed kettle of steaming water to which 2 cc ($\frac{1}{2}$ teaspoonful) of spirits

of camphor has been added. Expression of the blackhead with a comedone extractor then becomes easy. The nightly application for very short periods and in a well ventilated room of compresses of carbon tetrachloride is equally beneficial for the removal of blackheads.

The application of *adhesive plaster* for twenty-four hours over an area containing blackheads will often result in their extraction on the forceful removal of the plaster.

In order to effect the removal of blind comedones, the overlying epidermis must be incised with a scalpel.

In the comedone type of acne, treatment is designed to produce mild continuous exfoliation. This can be accomplished by the use of ointments or pastes containing sulfur resorcin, beta naphthol, or a combination of all of them. If the medication is dispensed in a paste it should be applied thickly at night, or if the medication is contained in an ointment it should be rubbed into the skin at night and the medication removed with soap and water in the morning.

The following ointment is mildly keratolytic

Resorcin	3.0
Ichthyol	5.0
Beta naphthol	2.0
Ung. aquaphor	4. 100.0
Sig. Apply 1 bedtime	

Another useful method of peeling the skin is with a slush, free of solid particles which may burn the skin, made by dissolving solid carbon dioxide in acetone. This therapy is applied according to the following plan: 100 cc. (3 ounces) of solid carbon dioxide are placed in a clay mortar and ground to a fine powder. Acetone is then slowly added while constantly stirring the mixture to obtain a smooth paste. 7.5 cc ($\frac{1}{4}$ ounce) of pec-

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Sodium arsenat	2.0
Phenol	1.0
Aq. dist.	q.s. d 100.0

M Dispense in a sterile rubber dam covered bottle.

Sig One drop subcutaneously. Increase the dose by 1 drop daily until 20 drops are given. Then reduce dose 1 drop daily.

Fowler's solution is of distinct value in acne conglobata.

Sutton and Sutton advise giving 2 grains of desiccated whole gland *thyroid* with the evening meal. The dose of thyroid must be adequate and just below the dose which produces symptoms of intoxication. After a few weeks the need for this dosage may no longer be present and the dose may then be halved or omitted.

Vaccines, bacteriophages, and toxoids are of little if any value; the occasional good results obtained by their use are no doubt due to nonspecific protein therapy.

The intramuscular or intradermal injection of *sterile milk adon* or the patient's own blood is sometimes helpful and should always be used in the indurated types of acne. *Iosterol* in doses of 20 000 to 100 000 units daily has been followed by improvement in many cases.

Istamm B complex increases the hydrochloric acid contents of the stomach and corrects constipation and is therefore helpful in treating acne. *Riboflavin* and *niacin* are also remedial measures.

Intravenous injections of *normal saline solution* or the oral administration of enteric-coated tablets of *sodium chloride* (15 gm every two or three hours) is valuable in treating pustular acne in which bromides or iodides are the etiological factor. Normal salt solution may also be employed with benefit if it is directly injected into the pustule and around the inflammatory base.

Tyrosine	30
Kali carbonate	10
Aq. benzoyl peroxide	63.0
Aq. camphora	25.0

Use. Apply on retiring.

Specific instructions on cleansing the skin are given to the patient. Patients with dry skins rarely acquire acne. When it occurs, they are instructed to clean the face with hot water and a super-fatted soap or to use the following emulsion

Menthol	0.25
Oil of rose	30.0
Sodium borate pulp	45.0
Aq. rose	1000.0

Use. The daily use of this lotion tends to soften the skin.

Cleansing the face with Castile soap or powdered almond meal is advised when the skin is delicate and not oily. The following formula is recommended instead of Castile soap when pustules are present.

Sulfur (oil-soluble)	10.0
Spts. rose oil	25.0
Bals. peru.	2.0
Camphora	2.0
Glycerin	2.0
Aq. rose, q. s.	60.0

Use. Apply to face twice each day.

Some patients with pustular acne do well when treated with tyrosinase, as follows:

Tyrosinase	0.003
Glycerin base	60.000

Use. Rub into skin three or four times daily.

In follicular pustular infections, the following is occasionally useful

Benzoyl peroxide	3.00
Glycerin ointment base	30.00
Hydroxyquinoline sulf.	0.63

Hot water and tincture of green soap are employed for the oily and indolent skin. Solvent, cleansing lotions containing alcohol, ether acetone, or carbon tetrachloride are valuable adjuncts in

treatment of acne associated with oily seborrhea. The following formulas are of value for oily and indolent skin

I

Ether or acetone	45.0
Sps. myrror or sp. odoratus q. s.	250.0

Use. Useful for removing oil from skin.

II

Thymol	0.25
Menthol	0.5
Ethyl alcohol	80.0

Use. To be applied to oily skin after washing in tincture of green soap.

III

Boric acid (colloidal)	4.0
Ether	12.0
Alcohol, q. s.	80.0

Use. Apply twice daily mopping affected areas with a sponge moistened with solution.

IV

Calamin. pul.	1.5
Zinc oxide pul.	1.0
Saps. soft	1.0
Oil. olive	16.0
Aq. rose, q. s.	80.0

Use. Useful for inflamed and tender oily skin.

V

Boric acid (colloidal)	2.0
Ether	1.0
Alcohol 70%	1.0
Mech. frag.	1.0
Liq. calc. hydraz.	15.0
Aq. rose, q. s.	80.0

Use. Indicated in acne associated with oily seborrhea.

Kummerfeld's lotion may be substituted for lotio alba according to the following proportions.

Camph.	1.0
Acet. pul. ben.	2.0
Glycerin	4.0
Boric acid (colloidal)	10.0
Aq. rose, q. s. ad.	100.0

M. Triturate finely powdered camphor with powdered wax and precipitated sulfur; then add glycerine and slowly add rose water triturating constantly to obtain homogeneous lotion.

Northrup states that, in mild cases of acne, a small amount of a saturated

precipitated sulfur is then added to the mixture, which forms a light yellow paste. An ordinary cotton pad is dipped into the slush and immediately applied evenly to prominent lesions with slight pressure. This procedure can be followed four or five times at the same sitting, one application being made directly over the preceding one. At each application there is a temporary blanching of the skin. This blanching is extremely evanescent and usually disappears after each application. Desquamation occurs the following day. The sulfur deposit of this mixture is allowed to remain on the face for twenty minutes and then washed off with cold water. Before resorting to this method of treatment and during the treatment the eyes are covered by wet boric acid compresses. Applications are repeated once a week until the desired therapeutic effects are attained.

This form of cryotherapy is not to be used after roentgen therapy without an interval of one month.

Lesions other than comedones should be left alone. Scarring often results from forceful manipulation by the patient. Dermal abscesses which do not involute spontaneously should be incised at their dependent portion and the cavity should be packed with gauze which is replaced every other day in order to make the lesions heal from within.

In cystic acne each sac must be punctured with a fine scalpel, and the contents gently expressed. Its disappearance is then hastened by x-ray therapy ($\frac{1}{4}$ erythema dose every five days for five doses).

LOTIONS. Acne pustulosa responds better to lotions than to pastes or ointments. In extensive pustulation *Lugol's solution* may be applied or *ammoniated mercury* in a greaseless cream base. The lotions in common use are *Vlemmick's*

solution, *lotio alba* and the many various other lotions containing sulfur. *Vlemmick's solution* is of special value in acne involving the back.

The same lotions are applicable to the other types of acne, though topical applications in these types are of less value than systemic treatment. The following lotions are of value in the chronic pustular type of acne.

Menthol	1.0
Sulfur (colloidal)	30.0
Sodium borate	8.0
Powder calamin	12.0
Acetone	45.0
Aq. camphorae, q.s.	240.0

The lotion is freely applied to the face two or three times each day. The skin will feel tight and rough and will peel within a week or ten days after using this lotion.

More conservative treatment is indicated for acute types of acne in which hyperemia is evident. Cipollaro found the following simple lotion very serviceable.

Camphor	0.10
Zinc oxide	4.0
Powder calamin	4.0
Magnesium carbonate	4.0
Glycerol	0.75
Aq. hamamelidis, q.s.	120.0

8cc. Apply several times each day for two weeks.

Freshly prepared *lotio alba* is applied after the inflammation has become less acute.

	A	
Zinc sulf		4.0
Aq. roseae, q.s.		60.0

	B	
Kal. sulfurata		4.0
Aq. roseae, q.s.		60.0

M. Combine A and B while stirring.

Blackheads can be successfully treated with the following lotion.

water and soap, and the lips protected by cold cream before employing keratolytic pastes. The paste is warmed and applied with a brush. The patient is usually given a preliminary test by employing the selected exfoliating past for a period of one or three hours. The paste is then applied for at least twelve hours when untoward effects are not evident. The red, inflamed face becomes brown and brawny after this application. The epidermis exfoliates in sheets after twenty-four to forty-eight hours application. Pustules disappear and superficial scars are reduced. This treatment is continued daily for a period of four days, or employed once each week for a period of five or six weeks.

The following preparations of keratolytics are highly recommended

I

Resorcin	40.0
Zinc oxide	70.0
Kaolin	8.0
Adeps ben	20.0

M. Tricretate resorcin 5th sorted benzoinated lard, then incorporate with oxide and kaolin.

II

Folkers (collidal)	21.0
Betaseph	10.0
Ether	10.0
Sapo med	24.0
Petrolat q	20.0

M. Mowles sulfur and betasephthol in ether and incorporate in soap and petrolatum.

III

Betaseph	10.0
Folkers (collidal)	20.0
Sapo med	20.0
Petrolat	20.0

Use Apply for period ranging from fifteen minutes to one-half hour the first day increase period of contact until patient can tolerate 24 hours

Peeling of the skin in acne scars may also be obtained by applying 20 per cent trichloroacetic acid at 5 minute intervals until the skin becomes white

Röntgen Therapy Röntgen therapy still holds a prominent place in the therapy of acne; however not every form of acne responds to the roentgen ray and other factors should be considered such as the texture of the patient's skin, its capacity for pigment production and the patient's age.

Röntgen rays should be used as little as possible and never without recourse to systemic measures. Röntgen therapy alone produces only temporary improvement and is promptly followed by recurrence whereupon the patient desires a repetition of the course. The result of several courses of treatment is excessive dryness, atrophy and telangiectasis. If other methods of treatment within a reasonable length of time show no improvement then only should we resort to roentgen therapy.

Acne patients with a relatively dry skin are a greater risk than those with an oily seborrhea. Blonde women with delicate skins, patients with rosacea, those with inflamed skins, and children are poor risks for roentgen therapy.

Cautious dermatologists should photograph the patient before starting roentgen therapy so as to have irrefutable proof of the amount of damage previous to treatment.

The best results are obtained by fractional irradiation with unfiltered rays in exposures of $\frac{1}{4}$ of an erythema dose (75 r) given weekly to the affected areas over a period of two months. The eyes, eyebrows, scalp, hair ears, and thyroid gland must be protected. The roentgen rays may also be adequately filtered through $\frac{1}{2}$ mm. of aluminum. Filtered rays are more beneficial in indurated acne vulgaris. All topical applications are avoided during the course of x-ray therapy.

solution of magnesium sulfate taken into the palm of the hand and rubbed gently all over the face until dry leaves a soft "bloom" on the face and has a beneficial effect in drying up the lesions

In cystic acne the top of the lesion should be removed with a trephine—the size varying with the lesion—and the contents expressed. The cavity is packed with narrow gauze and healing achieved by granulation.

THE SCALP. Treatment of acne must include treatment of the accompanying dry or oily seborrhea of the scalp. Often acne lesions on the forehead temples, and along the hairline or even elsewhere resist all treatment until the scalp and hair are properly cleansed and treated. The scalp should be shampooed weekly or twice each week if the hair is very oily and every fourteen to eighteen days if the hair is very dry. If the scalp is very oily a lather from sulfur soap should be employed and left on the scalp for half an hour. It should then be washed out and thoroughly rinsed with water to which the juice of a lemon has been added. Should the hair be dry the following solution should be added the evening prior to the shampoo.

Mineral oil	8.0
Castor oil	8.0
T. yoline	0.6

9. Heat and apply to scalp very hot and the following morning shampoo scalp with a shampoo made from Castile soap, or a liquid tar soap.

Daily applications of the following lotion are beneficial in seborrhea of the scalp

Hydrarg chlor corros.	0.05
Menthol	1.00
Resorcin monoacetate	8.00
Sp. formicarum	30.00
Oil myricae	2.00
Spt. vini rect, q. s. ad	250.00

Sig. Apply to scalp on alternative days. The application is best made with a medicine dropper employing a few drops for each area, and massaging it gently into the scalp with the finger tip.

Salicylic acid (4 gm) should be substituted in this preparation for resorcin monoacetate for individuals with light hair.

The following ointment is recommended for application to a dry scalp the evening preceding the shampoo.

Acid salicylic	1.0
Sulfuris (colloidal)	4.0
Ung. aq. rose	50.0

Sig. A shampoo with liquid tar soap should follow use of this ointment.

Biweekly applications of the following lotion are of value in the prevention of recurring seborrhea of the scalp.

Menthol	0.25
Acid salicylic	2.0
Hydrarg chlor corr.	0.05
Oil ricini	0.25
Sp. myrica or sp. odorat., q. s.	100.00

Sig. Apply several times each week.

Ointments containing 5 per cent ammoniated mercury or 10 per cent sulfonated oil applied to the scalp several times each week, are definitely beneficial in treating seborrhea sicca. It is essential to continue the treatment of dandruff for months because patients are often reinfected at their next visit to the barber or the hairdresser even when the condition does not relapse spontaneously.

EXFOLIATING PASTES. Such preparations as resorcin salicylic acid beta naphthol and sulfur are occasionally employed as keratolytics to remove the superficial layers of the skin. The underlying cutaneous structures remain unaltered when any of these keratolytics are carefully applied. Resorcin is probably the safest keratolytic to employ. The face is thoroughly cleansed in hot

skin, the absence of hair the localization of the lesions over the joints and extensor surfaces, together with the diffuse involvement and slight branny scaling differentiate this disease from a true

cum deposits, scleroderma, histiocytoma, lipoma, sarcoma and amyloid infiltration may also occur

Prognosis The disease is slowly progressive; however it may remain station



Fig. 14 *Acrodermatitis Chronica Atrophicans*. Note bladderlike lesions beneath skin of arm which, on pressure, can be inverted.

scleroderma. The skin in scleroderma is hard, smooth, boardlike, and closely adherent to subcutaneous tissue.

Complications Ulceration and carcinoma may supervene on atrophic patches, fibroma, pseudoxanthoma, cal-

ary for a long period. Complete involution never occurs.

Treatment Treatment is symptomatic and of doubtful value. Improvement may follow employment of vasodilators like acetyl-beta methyl-choline,

ACRODERMATITIS CHRONICA ATROPHICANS

SYNONYMS: *Diffuse idiopathic atrophy of the skin, atrophie maculosa cutis erythromelia, anetodermia*

Acrodermatitis chronica atrophicans is a chronic progressive disease characterized by diffuse atrophy of the skin principally affecting the extremities.

Varieties Two clinical stages are distinguishable (1) The primary stage of infiltration which leads gradually to (2) the stage of atrophy. Both stages are painless and the patient is often unaware of its development.

Incidence It occurs twice as frequently in females as in males and usually appears between twenty five and fifty years of age. The youngest reported case occurred at twelve years of age while the oldest was seventy years.

Etiology *Acrodermatitis chronica atrophicans* is rarely seen in American born individuals. It is commonly seen in the Rhineland and especially in the vicinity of Strassburg. Avitaminosis resulting from improper diet must certainly be considered as a possible etiological factor. Goiters also occur in these areas and Pautrier attributes the increased prevalence to thyroid pathology. Darier believed any of the following might be etiological factors: endocrine disturbance, tuberculosis, syphilis, leprosy, scleroderma, trauma and infections.

Pathology The early stage of inflammation is characterized by the absence of atrophy. The papillary portion of the corium is edematous and infiltrated with round and plasma cells, and the blood vessels are dilated. The elastic tissue finally disappears in some areas and becomes diminished or impaired in others. A distinct atrophy sets in.

The subcutaneous tissue is reduced and the glandular tissue gradually disappears. The epidermis is thinned out

to two or three layers of cells. The outstanding features of this disease are ulnar bands, fibrous nodules, scleroderma like changes, arthritis deformans of the hands, bone atrophy and ulceration over bony prominences, atrophy of the oral and vaginal mucosae and macular atrophy with ballooning. On the forearm the infiltration begins near the olecranon and follows the course of the ulnar nerve (so-called ulnar band or ulnar stripe). On the legs the infiltration begins near the patella and extends to the dorsum of the foot. These patients may also suffer from scleroderma like lesions on other parts of the body.

Symptoms The condition begins in variably on the dorsum of hands and feet and on the elbows as bluish red patches, gradually spreading to involve forearms and legs. The trunk is rarely involved. No cases have been reported with *acrodermatitis chronica atrophicans* of the face. Involved areas may be at first slightly edematous, scaly and occasionally weeping but remain generally in the same plane as the normal skin. Atrophy becomes manifest in the course of months or years, when the skin loses its elasticity and pliability becoming grayish, thin, shiny, wrinkled and lax. The skin does not spring back into position when pulled from the surface because of the loss of elasticity. This condition is called *anetodermia*. The subcutaneous tissue and occasionally the underlying bony structures are likewise atrophied. Hairs are absent and sweat glands are atrophied. Itching may be a pronounced feature in the late stages.

Diagnosis The age of the patient, the cigarette paperlike wrinkling of the

of the cord and brain show marked edema and congestion.

Symptoms. The two varieties of this disease have similar clinical features and the symptoms are identical. The common constitutional symptoms comprise irritability, fatigue and anorexia. Butler considers the pathognomonic symptoms resident in the feet and hands and consisting of edema, itching and pain. The character of the eruption appearing on the body varies. It may simulate the eruption of scarlet fever (German measles, urticaria, erythema and eczema, or it may be suggestive of anaphylactic dermatitis. Photophobia and laceration are often present. The disease has an insidious onset, either the nervous symptoms or the dermal symptoms may appear first.

The affected child becomes listless, complains constantly, has more or less insomnia, complains of thirst, refuses food, and loses weight. Generalized perspiration is present, more severe on the hands and feet which are cold and clammy. The palms and soles become erythematous, hence the name pink disease. A multiform spotted erythema involves the trunk and itching is very severe. Desquamation follows the rash and may continue over a period of many weeks. Deep muscle pain as well as pain in the abdomen have been reported.

Brantwaite has called attention to the presence of hypotonic muscles, mental depression, and the rapid eruption of teeth. Deciduous teeth are sometimes lost. The child walks with difficulty and prefers to lie on its abdomen, or crouched on the side with the face in the pillow. The pulse is rapid, systolic blood pressure and the basal metabolism rate are

increased. Feline disturbances are occasionally present.

The duration of the attack is from one to three months. Recurrences have been reported.

Diagnosis. It must be differentiated from infantile pellagra, ergotism, and ustilaginism (moldlike fungus). The lesions of acrodyma, consisting of photophobia, paresthesia, perspiration, peeling and "pinkness," are pathognomonic and prevent errors in diagnosis.

Complications. Multiple cutaneous abscesses, which are difficult to heal, occasionally occur. Bronchitis, pneumonia and pyelitis have been reported as complications of this disease.

Prognosis. Relapses may occur. Pneumonia causes a fatal termination in about 10 per cent of the cases.

Treatment. No unanimity of opinion prevails as to the best treatment. Fier recommended tropine in dosage of 0.001 to 0.002 gm. daily. Maklan Massot claimed a cure by adding to the diet vegetables, meat juice, lemon juice and liver together with vitamins A and D and thyroid and adrenal gland extracts.

In a recent case of pink disease in a child of nineteen months, a cure was effected after the daily administration of 50 mg. thiamin chloride (B_1) intramuscularly and pyridoxine (B_6) every other day intravenously. At the same time accessories was given by mouth.

Crawford recommended forced feeding with foods rich in vitamins A and B, abundant orange and tomato juice, yeast, and cod liver oil.

Locally antipruritic powders and lotions may be used. Measures must be taken to prevent the patient from scratching too vigorously.

The use of the *mercury quartz lamp* is soothing

Ferment Therapy Sells believes that the disease is due to ferment disturbances and considers ferment therapy effective. He recommends the following regimen of treatment

1 The daily intake of 100 to 200 gm ($3\frac{1}{2}$ to 6 3/4 ounces) of *raw pancreas* served at breakfast in warm (not hot) bouillon or mixed in potato puree.

2 The ingestion of tablets of *pancreatic ferment* containing amylase lipase and trypsin (holadin tablets) on an empty stomach. Two tablets are taken three hours before breakfast, one hour before lunch and one hour after dinner

3 Tablets of *intestinal ferment* containing duodenum are combined with the pancreatic ferment. Intestinal ferment is taken at bedtime

4 Tablets of *liver extract* and injections of liver extract are given with the pancreatic ferment treatment.

Injections of pancreatic extract (24 to 32 minims [$1\frac{1}{4}$ to 2 cc]) free from pancreatic ferment are administered two or three times each week in addition to ferment therapy

The condition usually progresses for several years and then becomes stationary

Sympathectomy Periarterial resection of the sympathetic nerve has been advocated. It has been tried by the author with benefit to the patient however after a few months the condition continued to progress. The Sells treatment has also been tried over a period of six months with the same result

Hormonal Therapy Treatment with thyroid posterior pituitary and suprarenal extract seems most logical

ACRODYNIA

SYNONYMS: *Swift's disease eryth ed m chelodalgia, erythroderma polynervitis pink disease trophodermato neonosis, Feer's disease pedionalgia epidermica.*

Acrodynia is a constitutional disease characterized by redness, edema itching and pain in the hands and feet. On the face and body an erythematous scarlatiniform or rubelliform eruption characterizes the disease. The constitutional symptoms are referable to the nervous system

Varieties Two varieties are described the *first* is the result of vitamin B₁ (thiamine) deficiency and the *second type* of acrodynia resembles ergotism and pellagra and is probably the result of improper food or some toxic agent

Incidence Children of either sex are equally affected. Most cases occur in the summer or autumn. It occurs more often between the third and forty-eighth months of life.

Etiology Some authors consider this disease the result of avitaminosis B₁, others regard it as an infectious disease some regard it as a toxic disease and a few believe it to be a virus disease. It is apparent that the cause is unknown

Pathology Warthin considers "that essential pathological changes in this disease are extreme edema and slight meningeal irritation of the central nervous system, chronic erythema of the skin, hyperkeratosis, hypertrophy of the epidermis and sweat glands and slight pigmentation of the rete occurring in children of the hypoplastic lymphatic constitution with associated or terminal respiratory infections and gastrointestinal catarrh and inanition. There is no evidence of polynneuritis. The meninges

of the cord and brain show marked edema and congestion.

Symptoms The two varieties of this disease have similar clinical features and the symptoms are identical. The common constitutional symptoms comprise irritability, fatigue, and anorexia. Butler considers the pathognomonic symptoms resident in the feet and hands and consisting of edema, itching, and pain. The character of the eruption appearing on the body varies. It may simulate the eruption of scarlet fever, German measles, urticaria, erythema, and eczema, or it may be suggestive of anaphylactic dermatitis. Photophobia and lacrimation are often present. The disease has an insidious onset, either the nervous symptoms or the dermal symptoms may appear first.

The affected child becomes listless, complains constantly, has more or less insomnia, complains of thirst, refuses food, and loses weight. Generalized perspiration is present, more severe on the hands and feet, which are cold and clammy. The palms and soles become erythematous, hence the name pink disease. A multiform spotted erythema involves the trunk and itching is very severe. Desquamation follows the rash and may continue over a period of many weeks. Deep muscle pain as well as pain in the abdomen have been reported.

Brathwaite has called attention to the presence of hypotonic muscles, mental depression, and the rapid eruption of teeth. Deciduous teeth are sometimes lost. The child walks with difficulty and prefers to lie on its abdomen, or crouched on the side with the face in the pillow. The pulse is rapid, systolic blood pressure and the basal metabolism rate are

increased. Febrile disturbances are occasionally present.

The duration of the attack is from one to three months. Recurrences have been reported.

Diagnosis It must be differentiated from infantile pellagra, ergotism and ustilaginism (moldlike fungus). The lesions of acrodynia, consisting of photophobia, paresthesia, perspiration, peeling and "pinkness," are pathognomonic and prevent errors in diagnosis.

Complications Multiple cutaneous abscesses, which are difficult to heal, occasionally occur. Bronchitis, pneumonia, and pyelitis have been reported as complications of this disease.

Prognosis Relapses may occur. Pneumonia causes a fatal termination in about 10 per cent of the cases.

Treatment No unanimity of opinion prevails as to the best treatment. Feer recommended atropine in dosage of 0.001 to 0.002 gm daily. Maldan Massot claimed a cure by adding to the diet vegetables, meat juice, lemon juice, and liver together with vitamins A and D and thyroid and adrenal gland extracts.

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Crawford recommended forced feeding with foods rich in vitamins A and B, abundant orange and tomato juice, yeast, and cod liver oil.

Locally antipruritic powders and lotions may be used. Measures must be taken to prevent the patient from scratching too vigorously.

ACROMEGALY

Acromegaly is a chronic disorder characterized by hypertrophy of the bones and soft parts, particularly of the face and extremities.

Incidence The age incidence is between twenty and thirty years. Males are usually affected. Females may be affected however they would be much older than males.

Etiology This disease has been reported among many races in various parts of the world. It is caused by hypersecretion of the anterior lobe of the pituitary gland. This may result from glandular hypertrophy which could be demonstrated by x-ray showing increased size of the sella turcica.

Pathology The characteristic skin changes include hypertrophy coarse and wiry hypertrichosis, and areas of hyperpigmentation. Epidermal glandular and collagenous hypertrophy and pigmentation of the rete are equally characteristic. The lips are thick and negroid in type and the tongue is hypertrophied. The hands and feet are generally enlarged.

Symptoms The onset of the disease is very insidious. The first symptom may be bilateral headache. Lassitude and loss of sexual power are early symptoms of the disease. The hands and feet enlarge and the height of the patient increases. The bones of the face enlarge more rapidly than those of the head.

The lower jaw is generally enlarged in nearly all the cases. The supraorbital ridge becomes very prominent, which feature is characteristic of acromegaly. The nose broadens and becomes coarse, the lips thick, and the lower lip often everts. The tongue hypertrophies and appears too large for the mouth. The skin becomes pigmented and hyperhidrosis and hypertrichosis are usually present. Body hair becomes coarse and profuse.

Diminution of the fields of vision is very common. In the beginning of this disease glycosuria is occasionally present. When the disease becomes stationary sugar tolerance is above normal. Blood pressure is abnormally low and the body temperature is usually subnormal.

The nails are often thick, flattened and longitudinally grooved.

Diagnosis This disease may be mistaken for osteitis deformans and pulmonary osteoarthropathy. In osteitis deformans there is no increase in stature. In acromegaly the injection of extract of the anterior lobe of the pituitary gland causes a rise of temperature. This phenomenon is not present in other conditions.

Prognosis The disease is either progressive or remains stationary after reaching a certain growth.

Treatment Treatment does not influence the course of the disease.

ACTINODERMATITIS

SYNONYMS Roentgen and radium dermatitis, xodermatitis.

Actinodermatitis is an inflammation of the skin resulting from exposure to certain amounts of roentgen or radium rays.

Varieties Two varieties are described the acute and the chronic.

Acute Actinodermatitis For descriptive purposes three degrees of acute dermatitis are noted (1) first-degree dermatitis (2) second-degree dermatitis, and (3) third-degree dermatitis.

Chronic Actinodermatitis. Chronic radiodermatitis may follow a first-degree burn if followed by frequently repeated exposures to a soft ray. It is, however, more likely to follow a second-degree burn and it invariably follows a third degree reaction. This form of radiodermatitis is usually encountered in physicians and technicians who are handling x-rays and radium. The sites of predilection are the knuckles and the flexor surfaces of the right thumb and

the protection of patients, physicians, and technicians have reduced the accidental "burns" which were so common during the early days of the roentgen and radium era.

Etiology The factors responsible for actinodermatitis include the quality of radiation, the dose administered, the interval between the various irradiations, and the patient's susceptibility. Carelessness is also an important factor and includes inadequate protection of opera-



Fig. 11 Roentgen-Ray Dermatitis. (Courtesy of Dr. Carroll S. Wright.)

index finger. The skin is dry thin and shiny. Itching and burning are the subjective symptoms and these are usually worse in winter. Freckles, diffuse pigmentation, telangiectasia, permanent alopecia, skin atrophy, keratosis, warty growths, and carcinomatous changes are characteristic of chronic radiodermatitis. Chronic radiodermatitis may develop as late as ten years, although the average is about eighteen months following dermal injury by x-ray or radium.

Incidence. Modern advancement in the calculation of roentgen and radium dosage and technical improvements in

tors and patient improper machine calibration, failure to note the time of exposure, omission of proper filters, too high initial doses, prolonged fractional irradiation, or too brief intervals.

Pathology The histological findings vary with the stage of the disease. In the acute form, both epidermis and cutis are affected, however the cutis shows the most important changes.

The primary change is a dilatation of all the vessels of the corium and particularly those of the upper network. Mild roentgen or radium doses cause a swelling of the endothelium of the cap-

illaries and lymph spaces. Stronger doses increase the changes and the epithelium becomes involved. Many of the cell nuclei are destroyed and clusters of chromatin granules are scattered in the involved cells. A very fine granular pigment can be found throughout the epithelium in the cells and the inter

tissue does not appear to be influenced. If destructive roentgen and radium doses have been applied ulceration and necrosis are produced.

In the chronic form of reaction the stages described under the acute form are present although the changes occur more slowly. Ulceration usually occurs



Fig 16 Roentgen-Ray Dermatitis.

cellular spaces. The pigmentation is most marked in the basal layer and appears in the form of a "distal pigment cap." The pigment is found only in the unaffected normal cells while the swollen and degenerated ones are free from it.

Larger doses are particularly destructive to hair papillae. Sebaceous and sweat glands undergo the same degeneration and pressure changes. The nerve

late Telangiectasia lentigo and warty growths are characteristic of x ray dermatitis.

Symptoms. The reaction following the use of x rays and radium are similar although the onset and course vary. These variations depend upon individual predisposing factors as well as the intensity and penetration of the radiation. Radium dermatitis depends upon the dosage. The beta rays of radium

cause a superficial dermatitis, while the gamma rays produce the same type of dermatitis as roentgen rays.

The acute reactions follow a single large exposure, or from multiple doses applied at frequent intervals.

First-degree reaction appears from two to seven days after exposure. It is characterized by burning and itching associated with erythema and slight edema. These symptoms usually disappear in

several days. The sweat glands and sebaceous and sudoriferous glands is temporarily impaired and the skin becomes atrophic and the seat of numerous telangiectasia. Freckling and keratosis usually follow in the course of several months. Severe tenderness and pain invariably accompany this reaction. The duration of second-degree dermatitis is from two to four months. Chronic changes usually manifest themselves later in life.



Fig. 17 Acute Roentgen-Ray Dermatitis.

the course of a week followed by scaling, temporary pigmentation, and slight dryness of the skin. There may be periodic recurrences of erythema which may be present for several days, although each recurrence is less intense than the preceding one.

Second-degree dermatitis is characterized by more pronounced erythema and edema, and occasionally by vesiculation. The reaction occurs several days earlier than those of the first degree. The hair of the irradiated area falls out in about three weeks and the resulting alopecia may be permanent. The nails may disquamate and the function of the sebaceous

The third degree radiodermatitis is the most severe and is characterized by the presence of necrosis and ulceration. Minor reactions of the skin may escape notice. Ulceration and necrosis may be the first sign of third-degree reaction although it is usually preceded by scarlet erythema, edema, vesicles, bullae, and sloughing. Intense pain is invariably an accompaniment of this degree burn which is intensified if cartilage, periosteum, or bone is involved. The time of onset depends upon the magnitude of the dosage and the softness of the radiation. With soft radiation, vesiculation precedes ulceration by a few

illaries and lymph spaces. Stronger doses increase the changes and the epithelium becomes involved. Many of the cell nuclei are destroyed and clusters of chromatin granules are scattered in the involved cells. A very fine granular pigment can be found throughout the epithelium in the cells and the inter

tissue does not appear to be influenced. If destructive roentgen and radium doses have been applied ulceration and necrosis are produced.

In the chronic form of reaction, the stages described under the acute form are present, although the changes occur more slowly. Ulceration usually occurs



Fig. 16: Roentgen Ray Dermatitis.

cellular spaces. The pigmentation is most marked in the basal layer and appears in the form of a "distal pigment cap." The pigment is found only in the unaffected normal cells while the swollen and degenerated ones are free from it.

Larger doses are particularly destructive to hair papillae. Sebaceous and sweat glands undergo the same degeneration and pressure changes. The nerve

late. Telangiectasia, lentigo and warty growths are characteristic of x-ray dermatitis.

Symptoms. The reaction following the use of x-rays and radium are similar although the onset and course vary. These variations depend upon individual predisposing factors as well as the intensity and penetration of the radiation. Radium dermatitis depends upon the dosage. The beta rays of radium

ACTINOMYCOSIS

SYNONYM *Lumpy jaw.*

Actinomycosis of the skin is a chronic inflammatory mycotic disease due to a vegetable fungus known as the Actinomyces bovis (ray fungus)

Varieties Primary actinomycosis of the skin is rare (1.0 to 3.0 per cent). Actinomycosis of the lung is rarely primary but usually due to extension from the mouth or pharynx. Primary actinomycosis of the intestinal tract constitutes 90 per cent of the total number of cases. The cecum, appendix, duodenum, and gallbladder have been primarily affected. The disease rarely attacks the bones and then only secondary to an intestinal or pulmonary infection. In the latter case, the vertebrae are usually involved in the process.

In addition to the *Actinomyces bovis*, which is the most frequent cause of human actinomycosis, there are other closely related *Streptothrices* which produce similar cutaneous and subcutaneous lesions. These cases are classified as *sarcoidosis cutis*, *trichinocardiosis*, and *torulosis*.

A variety of actinomycosis designated as *Madura foot* or *madurella* is occasionally seen in the United States, but it is predominantly a tropical disease. It usually involves the foot and remains localized.

Incidence Men are more commonly affected than females. Farmers, dairy men, and those in contact with domestic animals are more likely to contract the disease. It occurs more often between the ages of twenty and forty.

Etiology The *actinomyces bovis* is a saprophyte occurring in nature on straw, oats, wheat, barley and in dust, pollen, or chaff from grain stalks. They have been found in the soil and may

occur as saprophytes in the alimentary canal of larvae in tonsillar crypts, in carious teeth, and in the human gastrointestinal tract. The incubation period varies from a few days to several months. The history of human infection often reveals the habit of chewing contaminated straw



Fig. 18 Actinomycosis. Note "lumpy jaw."
(Courtesy of Dr. Henry H. Perkins.)

and grass. The disease is probably communicated to man by contact with infected animals. Actinomycosis is common in cattle, and in sheep, goats, and more rarely in horses and cats. The milk from diseased cattle is not infectious.

Pathology The histopathology is that of an infectious granuloma. Infection establishes a deeply seated nodular process accompanied by granulation tissue epithelioid cells, plasma and giant

hours or days. With hard radiation, necrosis is often the first symptom. Healing often requires months or years of treatment and in some instances, the ulcerations are so indolent that they never heal and are usually followed by cancer.

Diagnosis. The history of the case is usually diagnostic. The characteristic lesions with their sequelae make the diagnosis unmistakable.

Complications. Suppurative onychia, secondary infection, ulceration and carcinoma are the common complications of radiodermatitis.

Prognosis. The mild types of radiodermatitis usually resolve without permanent damage to the tissues. The prognosis of the third-degree and chronic variety is unfavorable.

Prophylaxis. Prophylaxis constitutes a proper calculation of roentgen dosage and the obtaining of history of previous radiations. Regions that have already been overexposed retain almost indefinitely an increased susceptibility to radiation as well as vulnerability to injury. The avoidance of irritating applications before during and after irradiation is necessary. This includes the avoidance of soap, water ointments and lotions.

Treatment. A first-degree radiodermatitis is treated with mildly astringent wet dressings, such as a 3 per cent boric acid solution, 1 per cent aqueous solution of aluminum acetate dilute lead water or buttermilk.

A second-degree dermatitis usually responds favorably to prolonged warm regional baths of potassium permanganate 1:15,000 a solution of warm nor-

mal salt, or a 1 per cent aqueous solution of tannic acid.

A third-degree reaction often reacts favorably to prolonged exposure to the steam douche. Good results have been reported following the daily application of Bier's hyperemia one to two hours at a time.

The use of the fresh whole leaf of *Aloe vera* is worthy of a trial in all stages of an actinodermatitis. The flat surface of the leaf is peeled off and the gelatinous inner substance is applied directly to the affected area. It should be renewed every two hours during the acute stage and every eight hours after the subsidence of the inflammation.

Ultraviolet ray irradiation or the use of the air-cooled quartz lamp is stimulating and may be used twice a week. The ulcers should receive a suberythema dose.

Dusting powder containing 10 per cent zinc perhydrol or 10 per cent sodium perborate is used with benefit, especially at bedtime.

Treatment of chronic ulcers is essentially surgical. Wide excision for large and electrodecaction for small ulcers give prompt relief of pain and produce more satisfactory results. Radon ointment is occasionally beneficial. Salves containing analgesics (such as benzocaine) are contraindicated since they often increase the size and depth of these ulcers probably because of the vascular constriction.

Warty formations should be excised or destroyed by electrodecaction. MacKee states that the beta ray of radium gives the best results in selected cases.

Diagnosis Actinomycosis is differentiated from scrofuloderma, sarcoma, carcinoma, blastomycosis, and tertiary syphilis. Diagnosis of actinomycosis is established by demonstrating the ray fungus in the discharge and in sections obtained by biopsy. In suspected lesions, repeated examination for the fungus is at times necessary.

Prognosis The prognosis is always guarded because it depends upon the involved area and the extent of the disease. If the disease is limited to superficial parts of the anatomy, recovery is the rule, especially when early energetic treatment is started.

Prophylaxis Careful examination of all domestic animals should go a great way in preventing this disease. Carious teeth and poor hygiene of the oral cavity should be corrected. The chewing of grass, straw or grains commonly found on a farm is interdicted.

Treatment The treatment of choice

for cutaneous lesions is excision. Large doses of potassium iodide by mouth are beneficial. From 3 to 8 gm (45 to 120 grains) of potassium iodide should be given daily for a period of a month or until toleration is reached. Three daily doses of $\frac{1}{4}$ to 1 grain of copper sulfate are indicated for individuals who cannot tolerate the iodides.

Myers reported good results from the combined internal and local use of thyroid.

The sulfonamides (sulfanilamide and sulfadiazine in particular) are of great value, especially if the lesions are deep-seated. Penicillin is also highly effective in actinomycosis and should be given to a total dosage of 5,000,000 to 6,000,000 units.

Local treatment consists of surgery and roentgenotherapy and radium. Applications of Lugol's solution, tincture of iodine, or 10 per cent pyrogallol ointment are helpful.

ADENOMA SEBACEUM

SYNOVIALIS Nervus sebaceus, steatodermoma, agglomeration vasculaire, adenoma of the sebaceous glands.

Adenoma sebaceum is a rare congenital affection consisting of small tumors originating from the sebaceous glands and usually occurring on the face.

Varieties Several varieties of adenoma sebaceum are recognized. (1) a prenatal form which is designated as *nervus sebaceus* (2) an acquired type which is referred to as *small sebaceous adenoma*, (3) a systemic prenatal type which is called the *Pringle type* of adenoma sebaceum.

Incidence Persons of defective mentality are most frequently affected. It is of common occurrence among epileptics. A history of the disease occurring in the family is often obtainable.

Etiology The cause is unknown.

Symptoms and Pathology *Prenatal Form of Adenoma Sebaceum.* This type of adenoma is either present at birth or appears within the first few months of life. The lesions consist of yellowish plaques which are usually located on the scalp, face behind the ears, or on the neck. The surface of these plaques is granular and shows patulous orifices of the sebaceous glands. The plaques may be solitary or in groups, and their distribution is either bilateral or unilateral. Subjective symptoms are absent.

Complications are uncommon, however in a small percentage of cases carcinoma occurs.

cells, and degenerative changes. The fungi can be demonstrated by biopsy taken from the edge of lesions. They are usually located near the center of a mass of granulation tissue consisting of polymorphonuclear leukocytes and epithelial



Fig. 19: Actinomycosis.
(Courtesy of Dr. Henry H. Perlman)

cells. Histocytes and eosinophiles occur at the periphery.

Symptoms. Cutaneous actinomycosis occurs about the regions of the mouth, thorax, and abdomen. The disease is frequently encountered about the face and neck secondary to involvement of the mouth. It may also appear about the cutaneous surface of the chest secondary to pulmonary actinomycosis. Cutaneous actinomycosis of the abdomen is secondary to intestinal infection. Cutaneous actinomycosis over the spine is secondary to vertebral involvement.

The earliest lesions are firm, purplish nodules which soften and break down forming sinuses which emit a purulent

discharge containing yellow granules consisting of masses of ray fungus. The involved area is usually limited, but the surrounding skin is adherent to the underlying markedly indurated structures. The development of lesions may extend over months or years. The tongue and carious teeth are common sites of actinomycosis. The subjective symptoms vary with the location of the disease. In those cases where the mouth is involved pain is experienced in mastication. Lingual involvement causes pain and inability to move the tongue. Pharyngeal involvement causes painful deglutition. Pulmonary involvement produces symptoms commonly seen in bronchopneumonia. The symptoms of intes-



Fig. 20: Actinomycosis. Not swelling and the excreted orifices of the subcutaneous sinuses.

tinal actinomycosis may simulate chronic appendicitis. Actinomycosis of the neck and mouth may continue for as long as eight years, while the pulmonary types rarely last longer than one year.

exist alone. Patients manifesting this complex of symptoms commonly die during the second decade of life in status epilepticus.

Microscopic examination reveals sebaceous adenomatosis with vascular proliferation and fibrosis.

The only possible complication is carcinomatous degeneration.

Diagnosis: This disease must be differentiated from molluscum contagiosum and epithelioma adenoides cysticum. The distribution of the lesion in adenoma sebaceum with a history of development in childhood and the characteristic histology of the lesions are diagnostic and should prevent error in diagnosis.

Sebaceous adenoma in the Negro is easily confused with dermatous papulosa

nigra (Castellani). This latter is a nevus condition, common and almost exclusive in Negroes, characterized by minute, discrete, round, usually multiple, skin-colored papules. The lesions are persistent, noninflammatory soft on palpation, with a smooth, sometimes wrinkled, surface and are located on the malar areas or on the cheeks below the eyes.

Prognosis. The tumors are harmless. Parents should be told that they occasionally become malignant and that mental deterioration may occur.

Treatment. The most efficient method of treatment consists of the destruction of the lesions by *electrodesiccation*. Therapy with *roentgen rays* and *radium* is of great benefit.

ALEPPO BOIL

SYNONYMS *Oriental boil, Bistara button, Delhi sore Kandahar sore Delhi boil, cutaneous leishmaniasis, Lahore sore.*

Aleppo boil is an infectious granuloma of the skin and subjacent tissues.

Varieties. Two varieties of this disease are known, namely an ulcerating and nonulcerating type.

Incidence. This disease is endemic in Asia Minor, India, and Northern Africa. The disease is also present in animals. In Persia, 40 to 50 per cent of dogs are affected with the disease. Several cases have been reported as occurring in the United States. Both sexes are equally liable to infection and no age is exempt.

Etiology. The cause of this disease is the *Leishmania tropica*. This is a parasite round or ovoid in shape, and about 2 microns long. The sandfly (*Phlebotomus papatasi*) is now generally supposed to be the host for this parasite. The affection is transmissible by direct or indirect contact and is autoinoculable.

It can be transmitted from animal to man. Incubation varies from a few weeks to six months. One attack confers complete immunity.

Pathology. The characteristic lesion is that of an infective granuloma which consists of granulation tissue with plasma cells, and large cells full of parasites. In the ulcerative type, the staphylococci are demonstrable and the parasites may be difficult to find, however they may generally be recovered from the margin of the lesion.

The histological picture is not diagnostic; it consists of a spheroid cellular infiltration which extends deep into the corium, and is composed of plasma cells, lymphocytes, endothelial cells, and polymorphonuclear leukocytes.

Symptoms. This disease is characterized by a slow-growing, indurated, indolent, itchy papule. This papule

The sebaceous glands show marked hyperplasia in microscopic section of the plaque

Acquired Adenoma Sebaceum. This is usually referred to as senile sebaceous adenoma. It occurs only in seborrheic persons. The lesions appear at the time of puberty and increase in number and



Fig. 21: Adenoma Sebaceum. The numerous pinpoint elevations over each cheek and the chin represent the end results of the partial removal by electrodesiccation of the adenomas present as long as the patient could remember. Concomitant malformations were the nasal deviation and a cartilage-like plaque in upper lip 1 ft wide both of which are evident in the illustration.

size as the patient becomes older. The lesions consist of one or several flat topped yellowish or purplish papules with a central pore which marks the sebaceous-gland orifice.

The histology shows an adenomatous hyperplasia of the sebaceous gland

Pringle Type The Pringle type of adenoma sebaceum is the most important type of adenoma which involves the face. The lesions are symmetrical and appear as tiny papules varying in size up to 5 mm and attain a height of 2 or 3 mm. Their color is pink or brownish red and in appearance they are waxy and translucent. A capillary telangiectasia is often associated with them. The lesions are either soft or hard, depending on the amount of connective tissue present in the lesion. The lesions occur on the cheeks, nose, and chin although papules are sometimes found on the mucous membrane, especially the gingiva.

In the Hallopeau Leredde type, the surface of the lesions is verrucous and histologically there is predominance of fibrous tissue. In the Balzer type the alterations histologically are chiefly in the hair follicles.

In addition to the facial lesions, soft fibrous lesions may occur over the sacro-lumbar region. These latter lesions show no adenomatous sebaceous glands but consist only of a fibrous tissue proliferation similar to the lesion of Von Recklinghausen's disease.

Adenoma sebaceum is often associated with tuberous sclerosis involving the brain mixed tumors of the kidney and subungual and perungual fibromatosis. According to Butterworth the dermatologic lesions associated with tuberous sclerosis consist in hyperplasias and tumors nevi disturbances of pigmentation changes in the nails, and hyperkeratoses. Rhabdomyoma of the heart myoma of the stomach and the uterus, and multiple tumors of the rectum have also been reported as occurring in this disease. The syndrome comprising mental deficiency epilepsy and sebaceous adenoma has been named "epiloia" by Sherlock. Any one of the triad may

exist alone. Patients manifesting this complex of symptoms commonly die during the second decade of life in status epilepticus.

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It can be transmitted from animal to man. Incubation varies from a few weeks to six months. One attack confers complete immunity.

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The histological picture is not diagnostic; it consists of a spheroid cellular infiltration which extends deep into the corium, and is composed of plasma cells, lymphocytes, endothelial cells, and polymorphonuclear leukocytes.

Symptoms. This disease is characterized by a slow-growing indurated, indolent, itchy papule. This papule

enlarges without ulceration unless secondarily infected. The number of lesions varies from one to a hundred; however, single lesions are rare; the number commonly seen are three to eight. The early lesions resemble mosquito bites which become reddish or livid in color and are covered with a fine adherent scale. The



Fig. 22. Cutaneous Leishmaniasis. Of three months' duration. In a girl nineteen years of age. The lesion is ulcerated. Note the bubonula-like nodules (lymphatic dissemination) around to the nasal lid of the lesion. (Courtesy of Dr. F. Sogher.)

papule, if only one enlarges until it reaches several centimeters in diameter. When there is more than one only the first papule enlarges while the others retain their original size. If the papule is not treated it enlarges and its covering scale becomes rupial in character. With out treatment the lesion undergoes evolution in about one year leaving a small superficial scar in case ulceration

has supervened a large scar with deformity may result. A generalized non-ulcerating form with papillomatous nodules occurring over the whole body has been reported. This variety appears chiefly on exposed surfaces.

Donovan bodies are found in the lesion in about 0.5 per cent of cases, although none are found in the blood stream. The lesions are not accompanied by adenopathy. Culture may be the only means of establishing the presence of parasites (Dostrovsky and Sogher).

The sites of predilection are the face and ears although the hands, feet, arms, and legs may occasionally be attacked. Constitutional symptoms are absent.

Histoplasmosis. During the past ten years, this infection formerly believed to be a rare tropical disease has been found widespread in the United States. It is characterized by irregular fever, emaciation, anemia, lymphadenopathy and splenomegaly. Small encapsulated organisms (the *Histoplasma capsulatum*) resembling Leishman-Donovan bodies, are found in the tissues and blood especially in the cells of the reticuloendothelial system. About 30 per cent of the patients show oral or cutaneous lesions (petechiae and ecchymoses, especially in the last few days of life), nondescript papules, and ulcers. Some are superficial but many are deep. In 20 to 25 per cent of the reported cases, infants under fifteen months of age were affected.

The diagnosis is made by finding the organism in the lesion in a biopsy specimen from the spleen or bone marrow in thick blood smears, or in blood cultures. The intradermal test with histoplasmin may be helpful although not specific.

Diagnosis. This disease may be mistaken for syphilis, yaws, lupus vulgaris,

and granulomas due to pus organisms. The diagnosis is established by the demonstration of the causative parasite—the *Leishmania tropica*.

Prophylaxis Avoidance of insect bites is advised. Proper screening of the sleeping quarters is essential.

Complications A common complication of the disease is Vincent's infection.

Prognosis This malady is self-limited. The prognosis is therefore good. Immunity follows healing of the lesions.

Treatment Andrews states that a single erythema dose of x-ray produces a cure within ten days in the majority of cases.

Castellani recommends injections of oleum phosphoratum into the nodules

and around it, in doses of 5 minims twice weekly. Tartar emetic given intravenously in a 2 to 4 per cent aqueous solution is effective. The initial dose is 0.3 gm. and the injections are given twice weekly until a total of 15 gm. of antimony tartrate has been given.

Solid carbon dioxide, 2 per cent tartar emetic ointment, methylene blue, iodiform, and salicylic acid are some of the many local remedies.

According to Berberian, subcutaneous injections of vaccines of killed cultures of *Leishmania tropica* (3,000,000 to 5,000,000 *Leishmaniae* per cc. given in 0.1 to 0.5 cc. doses biweekly) induces a focal reaction followed by rapid healing of the lesions.

ALOPECIA

SYNONYMS Baldness, calities, falling hair, Kahlbetti, Hoerschornad.

Alopecia is a general term signifying loss of hair which may vary from a slight thinning to complete baldness.

Varieties The varieties of alopecia include alopecia congenita (also known as alopecia adnata, hypotrichosis, or atrichia congenita), alopecia prematura, alopecia senilis, alopecia symptomatice, alopecia seborrheica, and finally alopecia areata.

Alopecia Congenita

This is an ectodermal defect which is characterized by partial or total loss of hair.

Incidence Males are affected more often than females, the ratio being 2 to 1.

Etiology The cause is probably a faulty development of the hair papillae, matrix, and follicle or a regressive change occurring after normal development has already taken place. The disease is often familial. Birth injuries

(following forceps delivery) may be a causative factor.

Pathology The pathology shows varying degrees of development of the papillae and follicle. This is shown either by total absence of the hair follicle, hair papillae, hair matrix, or hair shaft.

Symptoms The hair loss may be confined to the scalp or may include the entire body surface. Complete loss of hair is rare. Lanugo hair and sweat glands may also be absent. Dental aplasia, nail malformations, various neurologic anomalies, and epidermolysis bullosa may be associated with this condition (see also Congenital Ectodermal Defects).

Prognosis It is unfavorable. Hair occasionally grows for a short time but it falls out again. This condition may continue in cycles and end without having produced a permanent crop.

Treatment No helpful treatment has been found.

Alopecia Prematura

SYNONYM: *Alopecia hereditaria*.

Alopecia prematura is characterized by hair loss that occurs in recurrent waves beginning at puberty.

Incidence Alopecia prematura occurs usually between the age of twenty and thirty years. It is more common in males. It does occur in women, but it is seldom as extensive as it is in the male.

Etiology The cause of alopecia prematura is unknown. Poor scalp hygiene appears to play a role in the early appearance of the disease. A history of the same condition in the forebears is often obtainable.

Pathology The loss of hair depends not upon the primary destruction of the hair matrix or follicle but rather on the fact that the power of hair regeneration is exhausted. Hair follicles in the cutis show a thinning of the epithelial walls, the lumina are occupied by loosely lamellated masses of horn while the connective tissue sheath has developed in a wide band of scarlike connective tissue. The hair bulb and papillae have either completely disappeared or atrophied. The vessels are dilated; however, signs of inflammation are either completely lacking or are slight.

Symptoms The scalp shows hair loss while the beard hair remains normal. The hair loss is symmetrical and its course may be slow or rapid. The progress is sometimes delayed by the presence of lanugo hair. The appearance of lanugo hair precedes the baldness which involves the entire scalp excepting a fringe of hair at the sides and back of the scalp. The baldness of alopecia prematura has the same shining appearance as that of senile alopecia. Grayness of the hair seldom precedes alopecia pre-

matura, while in alopecia senilis this is the rule.

Complications Pityriasis and hyperfunction of the sebaceous glands are often associated symptoms.

Prognosis The outlook is unfavorable, however, the progress of the disease may be delayed by proper treatment.

Prophylaxis The wearing of hats during the hot summer months should be compulsory. The hair should be thoroughly dried after washing it.

Treatment Alopecia prematura should be treated continuously for at least six months. Seborrhea is treated when present. Tight fitting hats are avoided. Drugs like pilocarpine, camphor chloral hydrate, and cantharides are employed for their stimulating and rubefacient effect. Vitamin H complex may be helpful. The following may also be useful (P.R.B. in):

Resorcinol monoacetat	12.0
Castor oil	12.0
Sulkylic acid	.33
Spt. of formic acid	48.0
Oil bergamot	0.1
Alcohol	q.s. 240.0

The best treatment consists of massaging the scalp each day for fifteen minutes followed by ultraviolet light therapy. The scalp should be covered with petrolatum prior to treatment.

Alopecia Senilis

Alopecia senilis is a variety of alopecia which has its onset after forty five years of age.

Incidence Senile alopecia is usually limited to the male sex. When it occurs in women it appears at a later age than in the male.

Etiology Alopecia senilis is probably the result of senile scalp changes; however, some consider it to be the end result of an alopecia seborrheica.

Pathology The histological reports vary considerably. The outstanding pathological picture is an endarteritis of the large vessels. Michelson believes that this causes the atrophy of the epidermis and a shrinking of the cutis with atrophy of the hair follicle.

Symptoms This type of baldness usually begins at the upper parietal regions associated with thinning on the sides. Occasionally the entire scalp is involved. The hair loss is usually gradual and a downy growth may persist for many months after the disappearance of the pigmented shafts. Thinning of the hair in the axillae and pubic regions occasionally accompanies senile alopecia.

Diagnosis The appearance and course of alopecia senilis does not differ from the seborrheic, hypopituitary or premature forms of alopecia excepting in the age of onset.

Prognosis The prognosis is unfavorable.

Treatment Male hormones and vitamin therapy are recommended.

Alopecia Symptomatica

SYNOVYM *Alopecia areolata*.

This is an alopecia of known origin.

Varieties Two varieties are recognized. Sabouraud divides them into several types, modified as follows.

The Accidental Type This type producing alopecia symptomatica is the result of

- 1 Burns, scalds, roentgenotherapy, radium, or avulsion.
- 2 Favus and kerion.
- 3 Erysipelas, acne necrotica, staphylococcus infection, herpes zoster varicella, tertiary syphilis, or carcinoma.
- 4 Miliary (ectopic) alopecia.
- 5 Lupus erythematosus and lichen planopilaris (Graham-Little)

6 Alopecia limbaire frontale, monilethrix, or ichthyosis of scalp

7 Folliculitis epilans of Quinquand (acne decalvans)

8 Pseudopelade of Brocq (alopecia atrophicans)

9 Lupoid sycomis (Brocq) or ulerythema sycomiforme (Unna) (see p. 687)



Fig. 23 Cicatricial Alopecia. Due to lupus erythematosus. Note beards of seith lupus erythematosus at borders of the alopecic area. (Courtesy of Dr. Jacques P. Guéguen)

10 Alopecia indurata atrophica

11 Folliculitis depilans (Arnouzan and Dubreuilh)

12 Folliculitis nares perforans (Colver)

13 Perifolliculitis capitis abscedens and sufficiens.

The destruction of the hair follicle constitutes the pathology in cicatricial alopecia. In the Arnouzan Dubreuilh type the folliculitis is located on the thighs and legs. In lupoid sycomis, it is the bearded area that is involved. In perforating folliculitis of the nose, the vi-

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SYNONYM: *Alopecia hereditaria*.

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Incidence Alopecia prematura occurs usually between the age of twenty and thirty years. It is more common in males. It does occur in women but it is seldom as extensive as it is in the male.

Etiology The cause of alopecia prematura is unknown. Poor scalp hygiene appears to play a role in the early appearance of the disease. A history of the same condition in the forebears is often obtainable.

Pathology The loss of hair depends not upon the primary destruction of the hair matrix or follicle but rather on the fact that the power of hair regeneration is exhausted. Hair follicles in the cutis show a thinning of the epithelial walls the lumina are occupied by loosely laminated masses of horn while the connective tissue sheath has developed in a wide band of scarlike connective tissue. The hair bulb and papillae have either completely disappeared or atrophied. The vessels are dilated however signs of inflammation are either completely lacking or are slight.

Symptoms The scalp shows hair loss while the beard hair remains normal. The hair loss is symmetrical and its course may be slow or rapid. The progress is sometimes delayed by the presence of lanugo hair. The appearance of lanugo hair precedes the baldness which involves the entire scalp excepting a fringe of hair at the sides and back of the scalp. The baldness of alopecia prematura has the same shining appearance as that of senile alopecia. Grayness of the hair seldom precedes alopecia pre-

matura while in alopecia senilis this is the rule.

Complications Pityriasis and hyperfunction of the sebaceous glands are often associated symptoms.

Prognosis The outlook is unfavorable, however the progress of the disease may be delayed by proper treatment.

Prophylaxis The wearing of hats during the hot summer months should be compulsory. The hair should be thoroughly dried after washing it.

Treatment Alopecia prematura should be treated continuously for at least six months. Seborrhea is treated when present. Tight fitting hats are avoided. Drugs like pilocarpine, camphor chloral hydrate, and cantharides are employed for their stimulating and rubefacient effect. Vitamin B complex may be helpful. The following may also be useful (P R B m)

Resorcinol monooacetate	12.0
Castor oil	12.0
Salicylic acid	.25
Sol. of formic acid	48.0
Oil bergamot	0.1
Alcohol	qs 240.0

The best treatment consists of massaging the scalp each day for fifteen minutes followed by ultraviolet light therapy. The scalp should be covered with petrolatum prior to treatment.

Alopecia Senilis

Alopecia senilis is a variety of alopecia which has its onset after forty five years of age.

Incidence Senile alopecia is usually limited to the male sex. When it occurs in women it appears at a later age than in the male.

Etiology Alopecia senilis is probably the result of senile scalp changes however some consider it to be the end result of an alopecia seborrheica.

Sutton and Sutton believe it to be due to arterial sclerosis, which causes malnutrition of the hair.

FOLLICULITIS NARIS PERFORANS (CULVER) The affection is rare and limited to males. A persistent crusted pustule develops on one or the other side of the

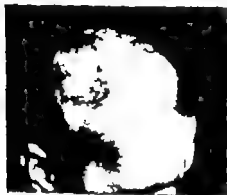


Fig. 26 Cicatricial Alopecia. Due to cicatrizing extensive basal cell cancer of ten years duration.

median line of the cutaneous surface of the nasal tip. The center of this lesion, after removal of the crust and expression of a droplet of pus, is found to contain a visible loose dead hair. Prompt healing follows removal of this hair.

According to Culver there is a primary folliculitis affecting the vibrissae within the nares. As the pustule dries intranasally the process deepens, the hair is devitalized and external perforation occurs. The dead hair is the cause of the persistent pustule.

PERIFOLLICULITIS CAPITIS ABSCISSUS AND SUPPURANS (HOFFMAN) Dissecting cellulitis of the scalp (Puscy) is a rare cicatrizing disease characterized by pea- or cherry-sized indolent nodules. These suppurate, sooner or later and there are fluctuating collection of pus in and beneath the scalp, especially in the occipital area. Sinuses and crust-covered areas

from the seropurulent discharge are present at the site of draining lesions.

The most permanent symptom is the undermining of the skin, permitting passage of a blunt probe from one involved area to another beneath the scalp.

Healing is accompanied and followed, by a temporary as well as a permanent localized cicatricial alopecia at the surface of the lesions.

A widespread pustular folliculitis and abscess formation, especially in infants, may simulate this condition although both belong in the group of chronic pyoderms with abscess formation.

Dissecting cellulitis of the scalp must be differentiated from perifolliculitis suppurative conglomerate (Lefloir) which is a deep-seated trichophytic infection



Fig. 27 Folliculitis Naris Perforans. (Courtesy Dr. M. B. Palmer)

characterized by boggy pseudocarbuncular lesions, often elevated and a follicular seropurulent discharge in which, over the affected areas, an alopecia of both temporary and permanent nature occurs.

brissae within the nose are involved. In this the infection extends deeply in volves the skin of the side of the nose, and perforates it. The pustule contains a destroyed hair.



Fig. 24: Alopecia. In Graham Little form of lichen plano-pilaris. There were on the trunk numerous typical papules of lichen planus as well as grouped areas of acuminate papules.

Most of the above forms are described elsewhere. The following are rather rare and merit discussion here.

ALOPECIA LIMBAIRE FRONTALE OF RABOURAUD. This form is characterized by baldness beginning in front of the ears. It commonly occurs in girls about the age of puberty. A history of crusting preceding the baldness is usually obtainable. The baldness is symmetrical and it spreads across the forehead to join the original preauricular areas. The anterior margin of the scalp is the only part affected. The baldness is permanent, however its progress may be arrested by using sulfur ointment. Sutton and Sutton consider this condition

an avitaminosis or endocrine affection.

ALOPECIA CICATHRICELLE MILIAIRE OF RABOURAUD. This form is characterized by large numbers of tiny scars scattered irregularly over the scalp. It occurs more often in aged scalps which are also seborrheic. It frequently involves the vertex. It is also more usual in women. It is commonly considered a milary form of acne varioliformis.

ALOPECIA INDURATA ATROPHICA. This form was first described by Pincus. It is a sclerosing form of scalp disease affecting the vertex, particularly of women.



Fig. 25 Folliculitis Depilans. Of limbe (Arnozan and Dubreuilh). Of several years duration, cleared up with hydroxyquinoline ointment. Pinpoint atrophic pits. A true cicatricial alopecia was the end result.

It causes cohesion of the connective tissue with the underlying aponeurosis which gives it a hidebound appearance. Diffuse baldness of variable degrees is present. It is often a familial disease.

Sutton and Sutton believe it to be due to arterial sclerosis, which causes malnutrition of the hair.

FOLLICULITIS NARIS PERFORANS (CULVER) The affection is rare and limited to males. A persistent crusted pustule develops on one or the other side of the



Fig. 26 Cicatricial Alopecia. Due to cicatrizing extensive basal cell cancer of ten years duration.

median line of the cutaneous surface of the nasal tip. The center of this lesion, after removal of the crust and expression of a droplet of pus, is found to contain a visible loose dead hair. Prompt healing follows removal of this hair.

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PERIFOLLICULITIS CAPITIS ABSCEDENS AND SUPPURIENS (ROFFAIAN) Dissecting cellulitis of the scalp (Pusey) is a rare cicatrizing disease characterized by pea to cherry-sized indolent nodules. These suppurate, sooner or later and there are fluctuating collections of pus in and beneath the scalp, especially in the occipital area. Sinuses and crust-covered areas

from the seropurulent discharge are present at the site of draining lesions.

The most permanent symptom is the undermining of the skin, permitting passage of a blunt probe from one involved area to another beneath the scalp.

Healing is accompanied and followed, by a temporary as well as a permanent, localized cicatricial alopecia at the surface of the lesions.

A widespread pustular folliculitis and abscess formation, especially in infants, may simulate this condition, although both belong in the group of chronic pyoderma with abscess formation.

Dissecting cellulitis of the scalp must be differentiated from perifolliculitis suppurative conglomerate (Lelorr) which is a deep-seated trichophytic infection



Fig. 27 Folliculitis Naris Perforans. (Courtesy of Dr. R. B. Palmer)

characterized by boggy pseudocarcinomatous lesions, often elevated and a follicular seropurulent discharge in which, over the affected areas, an alopecia of both temporary and permanent nature occurs.

It is a special form of kerion of Celsus (see page 751)

Treatment consists of surgical drainage and the swabbing of the cavity with 1 per cent tincture of iodine. Wet compresses of phemerol (1:1000) are bene-



Fig. 28: Syphilitic Alopecia. Occurs during the course of early (secondary) syphilis, usually a result of the toxemia.

ficial as are exposures to ultraviolet radiations. Intracutaneous injections of ascending doses of staphylococcus toxoid and the internal administration of cod liver oil are beneficial.

Noncicatrical Symptomatic Alopecia. This may result from the following

- 1 Mechanical causes.
- 2 Impetigo, impetigenous eczema, folliculitis, and tinea.
- 3 Trichotillomania
- 4 Toxic reactions produced by drugs such as thallium acetate, and boric acid
- 5 Alopecia following infection operation, childbirth fever and dentition in infants

6 Secondary syphilis, leprosy and tuberculous cachexia.

Sudden diffuse loss of hair may follow such systemic infections as typhoid fever, influenza, scarlet fever, pneumonia, and severe respiratory infection. It has also been observed during the puerperium. In carcinoma, tuberculosis and diabetes mellitus, the hair loss is much slower. Leprosy produces alopecia by leprosy infiltration of the follicle; however, the eyebrows and eyelashes are more apt to be affected. Endocrine disturbance may also produce hair loss. In pituitary disease, there is hair loss from the axillae, pubes, eyebrows, eyelashes, and the scalp (Simmonds' disease or pituitary cachexia). Hypertrichosis of the distal extremities and partial or complete alopecia of the vertex are characteristic symptoms of pituitary pathology. This type of alo-



Fig. 29 Syphilitic Alopecia.

pecia begins at the frontotemporal angle with gradual recession of the hair line along the temporal ridge, until finally the vertex is bald. Some hair growth is normally present along the lateral and marginal portions of the scalp. In Cush-

ing disease or pituitary basophilism, the hypertrichosis on the face and body is often associated with purplish lineal atrophæne. Hypothyroid baldness begins along the margin of the scalp, more often along the temporal regions, less frequently at the occiput and rarely extending to the vertex (Engelbach). Hypotrichosis of the eunuch or hypogonadal person does not involve the scalp. The scalp of a eunuch is usually covered with long coarse hair while in cases of acromegaly the hair is lost.

Alopecia Seborrheica

SYNONYMS *Alopecia pityriasis*,
alopecia furfuracea.

Alopecia seborrheica is similar to alopecia prematura excepting for the presence of seborrheic dermatitis (dandruff). Alopecia seborrheica is the most common type of alopecia.

This type of alopecia usually begins at puberty. It first involves the temporal and frontal regions and later the vertex. It is accompanied by furfuraceous desquamation and an itching which may be mild or severe.

Etiology: Alopecia seborrheica is caused by seborrhea. Seborrhea has usually been present for several years before hair loss is evident.

Symptoms. Each time the hair falls out, the recurring hair is thinner until finally nothing but downy hair remains in the follicle. The hairs are usually dry harsh, and lusterless, although rarely they may be excessively oily. The denuded areas slowly approach one another leaving nothing but a forelock in the middle of the frontal region and after the lapse of a few more years this, too, disappears and only a fringe of hair surrounds a bald scalp.

Treatment. The treatment and prophylaxis are similar to those of seborrhea and seborrheic dermatitis.

Alopecia Areata

SYNONYMS *Alopecia circumscripta*, *thinea decalvans*, *porrigo decalvans*, *area celsi*, *circular baldness*, *pelade*.

Alopecia areata is an affection of the hairy system characterized by rapidly appearing sharply defined bald areas, round, or oval in contour.

Varieties. Alopecia totalis and alopecia universalis are the varieties of alopecia areata.

In alopecia totalis, the entire scalp is bald, while in alopecia universalis the hair of the whole body surface is lost.

Incidence. It occurs chiefly between the ages of five and thirty and is rare after forty-five. The sexes are equally affected. The condition is more common during the fall, winter and spring months, and tends to improve in the summer.

Etiology. The etiology of alopecia areata has not been determined, however many predisposing causes have been recognized. The following are some of the predisposing causes: focal infection, endocrine disturbances, diseases of the nervous system, trauma, emotional or psychic shock, toxicity (thallium, arsenic or lead), infection with bacteria or fungi, epidemic diseases, heredity, certainly in some cases, angiospasm, and trophoneurosis due to some abnormality of the sympathetic nerves supplying the hairy parts or its vessels.

Bulzberger and Wolf believe sudden emotional or psychic shock is the most common predisposing cause and precedes the onset of hair loss. Instances are commonly seen where there has been a preceding shock to the nervous system.

Pathogenesis and Pathology. The pathogenesis of alopecia areata and alopecia totalis is a mystery. Seboraud's infectious and toxic theory as well as Joseph's trophoneurotic theory have

been discarded for the belief that the disease is due to a disturbance in the secretion of the ductless glands. We are indebted to Sabouraud for the report of most minute histological changes in the hair follicle in the acute stage. Under the term *urticule peladique* he described changes occurring in the upper third of the involved follicle namely between the follicular opening and the opening of the sebaceous gland into the follicle. The openings of these follicles are dilated and the surrounding tissue is erythematous and slightly edematous. The wall of the utricle is composed of flattened and atrophic cells with marked disturbance in the normal palisade arrangement of the basal row of cells. Sabouraud demonstrated clumps of micro-organisms in the early stage which he considered the cause of this disease. He believed that further pathological changes are due to the toxins already produced by these organisms.

Whatever the nature and cause of alopecia may be it certainly is a process that leads to atrophy. This is indicated by a lack of mitosis in the matrix with a gradual cessation of pigment formation. The shafts of the hair which normally have a smooth even gleaming surface, now appear rough splintered and nodular somewhat similar to the fibrillar decomposition seen in trichorrhexis nodosa. The root of this hair is normal. The pointed end corresponds to the completely atrophic hair bulb. Other hairs appear broken off just above the level of the scalp while toward the roots they appear pale and thin. The hair ends in undersized but normal roots.

In the early acute stage the root of the hair is involved, while in the chronic stage it is only that portion of the hair just above the root that is damaged (Unger)

It is evident that the intensity of the process is less than in the chronic stage. The toxins affect the hair papillae gradually and less intensely in this stage, and instead of causing its complete atrophy it causes a hypofunction of the papillae.

In this early stage, the superficial network of vessels of the corium is dilated and surrounded by a narrow mouth of small lymphocytes and occasional mast cells. Mast cells are only found in large numbers after the process becomes chronic. Large numbers of small lymphocytes may also be seen in the atrophic portion of the follicle above the sweat glands and in the space between the sebaceous glands and the hair follicle. After the actual loss of hair the inflammatory changes are more marked even if no causative agent can be shown. The deeper vessels are now also dilated and the infiltration is more marked. The infiltrating cells are also very numerous in the immediate vicinity of the follicle. The sweat and sebaceous glands in this stage are not involved. The collagenous and elastic tissues are normal. It is to be noted that in alopecia areata we find no band of hard almost scarlike connective tissue surrounding the hair follicle as is seen in alopecia seborrheica. If sections are cut many years after the onset of alopecia the picture is completely changed. In this case most of the follicles are dilated throughout their whole length and filled with masses of horn. The sebaceous glands have undergone a tremendous hypertrophy and open directly and vertically into the follicular spaces. The sweat glands remain normal. The normal papillary borders are retained although special stains show that the elastic tissue of the papillae has practically disappeared and that the collagenous tissue appears more ho-

mogenous. Deeper in the cutis the elastic fibers stain a bluish black instead of a brown with Weigert's elastic stain, they appear swollen and twisted. The connective tissue is sclerotic and the derma is often reduced to half its original thickness. With the exception of a few mast cells, all signs of inflammation have now disappeared. There is a complete absence of pigment in the surface and follicular epithelium.

eral may coalesce and form areas of wide extent. At the margins of the patches the hair may be painlessly extracted. On removal of these hairs one may notice an atrophy of the bulb; this gives the hair the shape of an exclamation point and they are called "exclamation point" hairs. Gradually these disappear and new hairs grow in the center of the patch. At first these new hairs are lanugolike and unpigmented but are



Fig. 30 Alopecia Areata. Associated with nail changes. Note transverse white stripes on fingernails.

In alopecia totalis the histological picture is similar to that of alopecia areata in the late stage.

Symptoms. Hair loss in alopecia areata is usually sudden and only occasionally is it gradual, in which event several days might elapse before baldness is noticeable. A typical patch of alopecia areata is sharply circumscribed, circular or oval in shape, glossy in appearance and completely devoid of hair. The areas of baldness enlarge peripherally and are

later replaced by stronger and pigmented hairs. Regrowth is slow and several crops of new hair may fall out before regrowth is permanent. The scalp is the site of predilection, although the bearded region, pubic region, axillae, eyebrows, and other parts of the body may be involved.

The skin of the bald spot is normal.

Ophiasis is a clinical type of alopecia in which the margin of the scalp is either partly or entirely involved. The baldness

progresses in a serpentine manner. It is most frequently observed in children. Associated disorders include leukoderma, nail changes, poliosis of the eyelashes, strabismus and other ocular symptoms. The nail changes are characterized by



Fig. 31: Alopecia Areata Universalis. Note in element of scalp, eyelashes, eyebrows, beard, pubes, axilla and chest. (Courtesy of Dr. Jacques P. Guequiere)

the appearance of transverse white stripes, punctate depressions, and brittleness. The more extensive the alopecia the more severe the nail changes and the poorer the prognosis.

Diagnosis. Circumscribed areas of hair loss with no broken hair fungi or scarring with inflammation and nor

mal skin differentiate alopecia areata from tinea, premature baldness, cicatricial baldness, lupus erythematosus, secondary syphilis, etc.

Prognosis. The average duration of alopecia areata is two years. The prognosis varies inversely with the age of the patient and the extent of baldness. In children and young adults, the prognosis is favorable. In patients over forty years of age and those with alopecia universalis, the prognosis is unfavorable. Treatment in all types of cases should be continued for at least one year before an unfavorable prognosis is given.

Treatment. Before commencing local therapy, the loose hairs on the border of the patch should be extracted; this can be done by grasping the hair between the fingers and exerting gentle traction. The local therapy should also be applied about $\frac{1}{4}$ to $\frac{3}{4}$ inch beyond the border of the patch.

Pure phenol lightly applied on the patch and wiped off with alcohol as soon as the skin turns white is recommended by Buckley and has proved serviceable in many cases. Other rubefacients in use include tar, mercuric iodide, tincture of iodine, chrysarobin, croton oil, colocynth, capsicum, salicylic acid, aqua ammonia, glacial acetic acid, cantharides, etc. Cutler's fluid, consisting of equal parts of tincture of iodine, phenol and chloral hydrate, may be used. This is applied to all parts once weekly or as soon as the irritation subsides.

The cause of alopecia areata being unknown, the treatment is empirical. Fortunately, most patients eventually recover without treatment; however, there is no doubt that treatment hastens recovery.

The maintenance of good health is an important factor in the treatment of alopecia areata. The principal remedies

prescribed in the constitutional treatment are arsenic, iron cod liver oil, pilocarpine hexamethylenamine thyroid extract, pituitary extract, gonads, suprarenal extract, foreign protein therapy, riboflavin, and vitamin B complex. Rest and change of locality are important in all cases. Sunlight, ultraviolet light (in graduated, ascending, stimulating doses) Grenz rays, x-ray (short of producing erythema) and high frequency sparking have all been recommended and are worthy of trial.

Foci of infection such as caries of the teeth, mouth abscess, diseased tonsils, chronic sinusitis, and impacted teeth must be eliminated. Regrowth of hair in areas long alopecic has been observed after the removal of impacted molars and of teeth having deep amalgam fillings.

Thyroid and arsenic are probably the most helpful remedies at our disposal.

In unilateral alopecia, the patient's morale will be helped by wearing a wig.

External treatment is an essential part in the management of every case of alopecia areata. The value of external treatment lies in the stimulation of the cutaneous circulation and of the nerve supply of the hair follicles.

Sabouraud recommends the following preparation in the early stage of the disease:

Acid acetici glacialis	10.0
Formaldehyd	1.0
Aq. calopurpurea, q	300.0
Rex. Apply daily. Rub stiff toothbrush on entire scalp. The following is to be applied to the areas of alopecia only.	
Spect. etheris composition (Lapque)	
(Hoffmann)	30.0
A. u. s. vertic. (crystal)	1.0
Rex. Apply daily by means of cotton pad.	

He advises the following ointment for more advanced cases.

Oil cedar (decolorized)	10.0
Tarpeth mineral (mercuric suboxide)	1.0
Petrolatum	10.0
Alipha. linae	10.0

Oil cerasa, q ad perfume

Rex. Ointment applied each night and scalp shampooed following morning in warm. Ointment applied three times each week and scalp shampooed once each in warm.

The ointment must be applied in small quantity and massaged into the entire scalp.

Sabouraud considers the following topical application the best medication. It is objectionable because it stains the integument.

Acid chlorophenici	30.0
(Chloroform)	30.0

Rex. Apply daily until marked local irritability occurs and integument assumes a mahogany color.

It is difficult to evaluate any form of therapy because of the high incidence of spontaneous cures in alopecia areata. The onset of recovery is heralded by the appearance of fine lanugo hair.

Alopecia Cicatricata

SYNOXIS Folliculitis decalvans, Quinquand's disease folliculitis epilans, pseudopelade (Brocq)

Alopecia cicatricata may be primary or secondary. The latter has been noted under Alopecia Symptomata (see page 8). Primary alopecia cicatricata is a rare form of folliculitis of the scalp characterized by the presence of erythema, pustules about the follicular openings, scaling hairless, smooth glistening and depressed scars.

Varieties. Besides military cicatricata alopecia and folliculitis depilans of Arnouzan and Dubreuilh two forms are described pseudopelade of Brocq and folliculitis epilans of Quinquand.

Pseudopelade of Brocq

Incidence. The majority of cases occur in males. Pseudopelade of Brocq

progresses in a serpentine manner. It is most frequently observed in children. Associated disorders include leukoderma, nail changes, poliosis of the eyelashes, strabismus and other ocular symptoms. The nail changes are characterized by



Fig 31: Alopecia Areata Universalis. Note in absence of scalp, eyelashes, eyebrows, beard, p. bcs, axillae, and chest. (Courtesy of Dr. Jacques P. Guequiere)

mal skin differentiate alopecia areata from tinea, premature baldness, cicatricial baldness, lupus erythematosus, secondary syphilis, etc.

Prognosis. The average duration of alopecia areata is two years. The prognosis varies inversely with the age of the patient and the extent of baldness. In children and young adults, the prognosis is favorable. In patients over forty years of age and those with alopecia universalis, the prognosis is unfavorable. Treatment in all types of cases should be continued for at least one year before an unfavorable prognosis is given.

Treatment. Before commencing local therapy, the loose hairs on the border of the patch should be extracted; this can be done by grasping the hair between the fingers and exerting gentle traction. The local therapy should also be applied about $\frac{1}{4}$ to $\frac{1}{2}$ inch beyond the border of the patch.

Pure phenol lightly applied on the patch and wiped off with alcohol as soon as the skin turns white is recommended by Buckler and has proved serviceable in many cases. Other rubefacients in use include tar, mercuric iodide, tincture of iodine, chrysarobin, croton oil, coloevuth, capsicum, salicylic acid, aqua ammonia, glacial acetic acid, cantharides, etc. Cutlers' fluid consisting of equal parts of tincture of iodine, phenol and chloral hydrate may be used. This is applied to all parts once weekly or as soon as the irritation subsides.

The cause of alopecia areata being unknown, the treatment is empirical. Fortunately, most patients eventually recover without treatment, however, there is no doubt that treatment hastens recovery.

The maintenance of good health is an important factor in the treatment of alopecia areata. The principal remedies

the appearance of transverse white stripes, punctate depressions, and brittleness. The more extensive the alopecia, the more severe the nail changes and the poorer the prognosis.

Diagnosis. Circumscribed areas of hair loss with no broken hair, fungi, or scarring with inflammation and nor

opy is valuable and temporary epilation may be necessary to avert its progress. Dr. Joseph Goodman suggests giving from 60,000 to 800,000 units of vitamin A daily. Locally 15 per cent sodium in goose grease 25 per cent ammoniated mercury ointment, or 10 per cent colloidal sulfur in petrolatum is occasionally beneficial.

Antiseptic lotions such as the following may be used with benefit

I	
Hyd. chlor. corr.	9 16
Acid salicylic	4 8
Ol. ricini	2 4
Sp. vin. rect.	100 0
Aq. kamamel.	250 0
II	
Acid salicylic	4 8
Sol. phenol. (1 1000) q	250 0

The disease runs a course of exacerbations and remissions; however it finally ends in permanent baldness.

AMYLOIDOSIS CUTIS

Systemic amyloidosis, as a pathologic febrile state occurring in the presence of chronic syphilis, visceral tuberculosis, and chronic visceral suppuration, has been known for many years. However it has been shown that in about 60 per cent of patients with amyloidosis, amyloid is present in skins that are clinically normal. Only in recent times have atypical systemic amyloidosis and primary cutaneous lesions, not associated with the factors found in so-called "secondary" systemic amyloidosis, been recognized. Clinically they vary considerably.

The formation and deposition of amyloid is unexplained in both the secondary and primary types. Amyloid is not a single substance however. It consists of protein and polysaccharide fractions (Hass).

For practical purposes, Michaelson and Lynch have classified amyloidosis cutis as follows.

- 1 Generalized secondary amyloidosis with cutaneous involvement
- 2 Systematized primary amyloidosis with maculopapular lesions
- 3 Localized amyloidosis
 - (a) Secondary type
 - (b) Primary or independent type

Generalized Amyloidosis with Cutaneous Involvement Cutaneous involvement in secondary or generalized

amyloidosis is rarely observed. In this form of amyloidosis, the contributing factors appear to be tuberculosis or prolonged suppuration. The usual sites of amyloid deposition are the liver, spleen,



Fig. 82 Amyloidosis, Tongue and eyelids. Part of systematized involvement. Progressive enlargement of tongue one year after onset. At first the tongue was lightly enlarged, covered with small translucent papules and localized purpuric spots. Later the tongue became much larger, deep furrows developed, and small areas became denuded and covered with pyogenic membrane as result of injury. At the end, the tongue was twice its normal size. (Courtesy of Dr. F. W. Lynch.)

affects children and young adults, while folliculitis epilans of Quinquaud develops in the fourth decade of life.

Etiology The cause is unknown. Hemolytic staphylococci are often found upon culture. Dr. Joseph Goodman of Boston expounded the theory that faulty metabolism of vitamin A was present. The course of the disease is extremely slow and is usually found to be progressive.

Pathology Microscopic examination of tissue reveals perivascular inflammatory changes. The infiltrate surrounding the lower half of the sheath of the affected follicle consists of polymorphonuclear leukocytes, a few plasma cells, and mast cells. The papillae are flattened and contain pigment cells. The sebaceous glands are atrophied. The stratum corneum is slightly thickened, the prickle layer is thinner and the granular layer entirely disappeared.

Symptoms Pseudopelade begins gradually, the first visible symptom consisting of erythema surrounding a hair follicle. A typical suppurative folliculitis is never found. The hair falls out and a minute scar is the result. Subjective symptoms, except for occasional pruritus, are absent.

Early cases resemble exceedingly mild sycois vulgaris of the scalp but as the disease progresses only the margin of the white scarred patches shows signs of inflammation. The picture comprises many areas of smooth thin atrophic glistening slightly depressed areas with whitish floor and pink, slightly elevated borders of irregular or angular outline. Tufts of hair may remain at the borders of the bald areas. The extracted hair reveals a succulent, white glassy sheath. There is no broken or exclamation point hair present in this disease. Common sites

are the vertex of the scalp and superior part of the occiput.

Folliculitis Epilans of Quinquaud

The diagnostic lesion of this variety is a reddish pinhead-sized inflammatory papule or pustule located at the orifice of a hair follicle and pierced by a hair. Crusting soon occurs and usually within a few weeks an atrophic bald spot results. This is a much more inflammatory condition than the pseudopelade of Brocq.

In both of these varieties, the follicular destruction in the involved area is complete and hair regrowth never occurs. While the scalp is the usual location of the disease, the bearded region is occasionally involved.

Diagnosis The disease must be differentiated from alopecia areata, favus, morphea, scars resulting from burns, and lupus erythematosus. In alopecia areata there is no bordering inflammation and scars are absent. In lupus erythematosus the follicular orifices are patulous, the borders of the area are often erythematous, and the cicatrized patches are infiltrated and covered with a grayish adherent scale. Patches of lupus erythematosus are often present elsewhere on the body, especially on the ears. In some cases, the diagnosis is impossible. In favus, the achorion schönlleini is found and the scars have no inflammatory border. Burns and scars from trauma are diagnosed by the history and inflammatory borders are absent.

Prognosis The alopecia resulting from this disease is permanent. The condition may last for many years, or the progress of it may be spontaneously arrested.

Treatment In mild cases ultraviolet radiation is beneficial. Roentgenother

in such lesions as naevi, senile keratosis, epitheliomas, cylindromas and some chronic dermatoses as secondary deposits.

Primary Type (*Lichen Amyloidosis*, *Amyloidosis Cutis Nodularis* and *Disseminata*, *Amyloidosis Cutis Lichenoides*) Although classified as a primary form of amyloidosis cutis, lichen amyloidosis is believed by some to be secondary to a previous inflammatory process. In any case, this form appears to be the one with fairly fixed clinical and histological characteristics. Primary cutaneous amyloidosis that is without amyloidosis of any of the viscera occurs but is extremely rare. It is chronic, benign, and usually observed in people in apparently good general health. The lesions are not always lichenoid, and the varied clinical pictures defy classification. In the lichenoid type, the lesions consist of intensely pruritic papules, usually localized on the extremities, especially the legs and forearms. The elementary lesion appears to be a pinpoint papule which is commonly brownish, sometimes translucent or pink; it is firm, with a slightly scaly surface. Papules may become so numerous as to form extensive infiltrative scale covered plaques, in

which nevertheless the papules can be easily palpated.

Diagnosis Lichen amyloidosis is commonly confused with lichen simplex chronicus (Widal). Lichen planus simplex and obtusus prurigo and nodular or giant lichenification. Amyloid may be detected histologically and by the Nomland vital staining method. Amyloid histologically stains bright red with dilute methyl violet, clear yellow with van Creson's solution, and dark brown with iodine. It is found deposited on the papillae just under the epidermis which shows a hyperkeratotic dyskeratosis. The vital staining test consists in injecting 1 cc. of a 1.5 per cent solution of congo-red intracutaneously in the vicinity of the suspected lesion. The papule if amyloid is present, becomes rose-red within twenty-four hours as the initial surrounding redness of the diffused dye fades.

Treatment This is often intractable. Phenol-containing lotions will help the pruritus. The x-rays have been of benefit in some cases. Fowler's solution in ascending doses has been of occasional value.

ANGIOKERATOMA

SYNOYKIS Telangiectatic wart, keratoangioma, lymphangioma, verruca telangiectatica, tuberculoid angiomastruma.

Angiokeratoma is a rare affection of the skin characterized by telangiectasis and pinhead vascular growths the surfaces of which are hyperkeratotic.

Incidence Angiokeratoma usually attacks young adults and rarely those past middle life. Females are more often affected than males.

Etiology The cause of this affection is unknown. Circulatory weakness, as evidenced by varicose veins, varicose veins, as well as chilblains, and lupus pernio

constitutes a predisposition to the disease. A hereditary tendency is occasionally noted.

Pathology Wile and Bielow state that the characteristic histology is primarily an injury to the blood vessel resulting in subepithelial and intraepithelial hemorrhage, epithelial proliferation, fragmentation, loss of elasticity and perivascular inflammation.

Symptoms Angiokeratoma often follows one or more attacks of chilblains.

and kidney. In Madden's case, a firm nodular swelling which proved to be due to amyloid deposition, developed three weeks before death.

Systematized Amyloidosis with Mucocutaneous Lesions. This term used by Iulbarsch designated a rare syn-

drome based on a diffuse, widely disseminated, but not generalized amyloid deposition. In this primary form of amyloidosis, the deposition occurs throughout the body, especially in the skin and musculature (lingual, skeletal, and myocardial muscles). These patients—in addition to pains and aches in the back produced by marked amyloid changes in

the skeletal muscles; purpura lesions, especially on the tongue, buccal mucosa and extremities, and a progressive downward course—present a painful glossitis with macroglossia with or without a cutaneous eruption of flat topped or spherical, smooth and firm papules of a waxy color



Fig. 33 Localized Lichen Amyloidosis. *Left* Showing pinpoint-sized lesions at the upper part. In the region of the wound, 1 ft. by removal of tissue for biopsy are a group of darker papules as a result of local staining with Congo red. *Injected locally.* *Right:* The papules have merged to form infiltrated, branny scaling brown patches in which the papules are still distinct. These patches simulate lichen simplex chronicus. There was intense itching. Local Congo red test gave selective staining of amyloid nodules a few away a 6 cm. (Courtesy of D. R. Nomland.)

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The lesions form plaques on the hands, but especially around the eyes, nose, mouth, and mucocutaneous junctures. The cause of primary systematized amyloidosis is unknown. Bunting and MacDonald relate it to Bence-Jones proteinemia and multiple myeloma.

Localized Amyloidosis. *Secondary Type.* In this type the amyloid is found

Intestinal Anthrax: This form is rare in man. It usually follows the ingestion of spore-containing meat. The symptoms comprise malaise, nausea, persistent vomiting, abdominal pain and tenderness and bloody diarrhea. Death occurs in three or four days.

Cutaneous Anthrax: Cutaneous anthrax is the result of direct inoculation by the anthrax bacillus. The sites of predilection are the face, neck, arms, and shoulders. The incubation period is short, ranging from a few hours to

case that it does not cause apprehension or distress in the patient, the patient's mind remains unperturbed until death.

Incidence: Anthrax is very common in Russia, China, Turkey, South Africa, and South America. It is less prevalent in the United States.

Etiology: Anthrax is primarily a disease of animals, usually sheep or cattle produced by the *Bacillus anthracis*, which is a gram positive encapsulated, nonmotile, spore-forming bacillus. It is conveyed to man by direct contact with



FIG. 33 Anthrax. Left: Eyelid. Right: Upper anterior chest, seventh day (Courtesy of Dr. A. C. LaBaccetta.)

several days. A vesicle or bulla preceded by an erythematous papule develops at site of inoculation which after the lapse of a day or two is filled with hemorrhagic serum. The vesicle or bulla finally ruptures and exposes a black tough eschar surrounded by a ring of smaller vesicles which is the so-called malignant pustule. A nonpitting, surrounding edema is present and the regional lymph nodes are enlarged and painful. Constitutional symptoms depend on the severity of the infection. In mild cases they may be entirely absent. In severe cases the symptoms are those of septicemia, namely: high temperature, rapid pulse, and early collapse. It is characteristic of this dis-

infected animals, or their carcasses, and by indirect contact through hides, wool hair or shaving brushes.

Anthrax is an occupational disease occurring among ranchers, shepherds, tanners, and woolsorters. Shaving and tooth brushes, contaminated with the spores of anthrax have been known to have caused the disease.

Pathology: The early lesion examined under the microscope shows vascular dilatation and marked interstitial edema with fibrosis network in the connective tissue. This network may also extend into the subdermal tissues and the muscle. Leukocytes are abundant in the edematous tissue, but remain in

Patients often suffer from cold, cyanotic hands and feet. Telangiectases or minute vascular dilatations are the first objective symptoms. They are pinhead in size, discrete, or grouped. At first they have a



Fig. 34: Scrotal Angiokeratomata of Fordyce (angiomas of scrotum) (Courtesy of Dr. Carroll S. Wright)

pink color later becoming a dark purple. The center of the lesion is darker than the remainder. After a short time these areas of telangiectasis become elevated, hyperkeratotic and assume a warty appearance. These points often coalesce

and form elevated papules or nodules, and the color becomes a dark red or purple. The sites of predilection are the dorsal aspects of the fingers and toes but the palms, soles, ears, and elbows and knees are occasionally involved. Subjective symptoms are absent. Lesions bleed readily if they are traumatized.

Diagnosis The primary pinpoint telangiectasis and subsequent hyperkeratotic tendency with the associated dark red or purplish color together with a history of chilblains, are diagnostic. The lesions are differentiated from verrucae vulgaris by the telangiectasis.

Furthermore, a history of trauma especially frostbite or exposure to cold resulting in injury of the blood vessels, is often obtained.

Angiomas of the scrotum (scrotal angiokeratomas of Fordyce) must not be confused with and are not related to angiokeratoma (according to Hudelo Wile, and others) although their surfaces are sometimes hyperkeratotic. They are real vascular nevi unassociated with injury, perivascular inflammation or hemorrhage (Wile).

Prognosis The lesions never disappear and there is no tendency to involution.

Treatment The lesions are readily destroyed by the *galvanic needle*, *electrocoagulation*, *solid carbon dioxide* or by *unfiltered radium*.

ANTHRAX

SYNONYMS *Malignant pustule*, *charbon*, *woolsorters disease*, *plenic fever*

Anthrax is an acute infectious and contagious disease of animals and man caused by the *Bacillus anthracis*.

Varieties Anthrax occurs in three forms.

Pulmonary Anthrax. Ragpickers and woolsorters usually develop the disease

by inhalation of the spores from contaminated hides or rags. The usual symptoms comprise extreme prostration, rapid respiration, feeble and rapid pulse, temperature 102 to 103 F., cough, and blood stained sputum. Death usually results in from one to five days.

It rarely follows the prolonged use of argyrol, protargol, and silver arsenaphena mine.

Localized argyria may result from the local use of silver following either topical application of nitrate of silver or the use of colloidal silver in the treatment of diseases of the eye, oral, and nasal passages, and the genitourinary tract. Conjunctival argyria may be part of generalized argyria or it may be purely local. The conjunctiva is usually brown in color gradually turning to slate or bluish-black. The hair and the nails may also be discolored, the hair having a faint red tinge.

Symptoms. The first signs of pigmentation appear on the edges of the gums. The discoloration may involve the entire cutaneous surface as well as the mucous membranes and internal organs.

Pathology. In cases of argyria silver occurs in the skin as an albuminate. The pigment occurs as a reduced silver in all parts of the skin except the rete

cells and the glandular epithelium. It is found in greater abundance just below the rete in the uppermost papillary layers of the corium and in the membranæ propriæ of the sweat glands. The elastic tissue is the most extensively involved. A deposit of silver is also found in the internal organs but it does not occur in the central nervous system.

Differential Diagnosis. Argyria must be differentiated from cyanosis, chrysiasis (gold pigmentation) and melanosis. Differentiation is made clinically and by microscopic examination. Silver is recognized as brownish granules in sections stained with pyronin methyl green.

Prognosis. The discoloration is usually permanent however the degree of pigmentation lessens in the course of many years.

Treatment. Some authorities advise the intradermal injection of an aqueous solution of 1 per cent potassium ferricyanide and 3 per cent sodium thiosulfate. Treatment has not been very satisfactory.

ATOPIC DERMATITIS

SYNONYMS. *Atopic eczema, infantile eczema, disseminated neurodermatitis, lichen chronicus simplex disseminatus, prurigo (Dowder) early and late exudative dermatitis (Ross), lichenified eczema, hay fever eczema, seasonal eczema.*

Atopic dermatitis is a dermal manifestation of idiosyncrasy or hypersensitivity characterized by the presence of any one or all of the primary skin lesions.

Varieties. Three distinct types are described, namely: the infantile, childhood or adolescent, and adult forms.

Incidence. Atopic dermatitis is one of the common varieties of skin disease. It is seventh in order of frequency in patients who are less than twenty-five years of age.

Etiology. Atopic dermatoses are caused by cutaneous hypersensitivity to environmental contacts, idiosyncrasies

in diet, or atopic diseases of other organs.

Exacerbations of atopic dermatitis may be caused by any of the following factors.

1. Heat or cold, or rapid changes in temperature.
2. Excessive perspiration.
3. Ingestion of certain foods or drugs such as fish, eggs, wheat, milk, chocolate or cocoa, salicylates, barbiturates, and the sulfa group of drugs.
4. Specific articles of clothing such as silk, wool, and nylon.
5. Exposure to dust, either house or factory.

small areas so that suppuration does not occur. The bullae are formed by multi-follicular collection of fluid with degeneration of the epithelial cells. Anthrax bacilli are easily seen in the border of the lesion.

Diagnosis. The characteristic features of anthrax are pathognomonic. Carbuncles, boils, smallpox, vaccination chancre and chancroid present similar appearances but the characteristic malignant pustule of anthrax is diagnostic. The *Bacillus anthracis* is demonstrable singly and in pairs in smears obtained from the pustule or the secretion of the lesion.

Complications. The usual complication is septicemia.

Prognosis. Anthrax is a grave disease but early recognition and prompt treatment should be effective. The pulmonary and intestinal types of anthrax inevitably end in death.

Prophylaxis. The carcasses of infected animals should be burned. All animals should be excluded from infected areas and should be immunized against anthrax infection. All hides, hair, wool and other materials which may be infected should be disinfected. Boiling of shaving brushes for three hours, or immersing them for four hours in 10 per cent formaldehyde solution at 110° F. is recommended in those cases where the origin of the brush may be from infected areas.

Woolsorters should wear masks and not eat in workrooms. Incineration of excretions of anthrax patients, and the

cremation of persons who have died from anthrax infection should be mandatory.

Treatment. *Antianthrax serum* should be injected locally as well as intravenously. The local injection of 10 cc. of antianthrax serum into the subcutaneous tissue surrounding the anthrax lesion completely circumscribing the lesion is the best method of treatment. These injections should be repeated every eight hours.

Systemic Treatment. Blood cultures should be made frequently to determine the presence or absence of septicemia. If septicemia is present from 200 to 500 cc. of antianthrax serum (being governed by the severity of symptoms) is given intravenously every twelve to sixteen hours for three doses; thereafter intramuscular injections of 30 cc. are given every twenty-four hours.

If septicemia is present give 200 cc. of antianthrax serum every three hours until the disease is controlled. Solutions of 1 per cent aluminum acetate or hot boric acid compresses afford local relief.

A combination of *antianthrax serum* and *neocarsphenamine* in full doses for three or four doses is considered the treatment of choice by some authors. *Sulfanilamide* should be given conjointly with the above-mentioned treatment if septicemia is present. *Penicillin* favorably influences the course of anthrax (it is practically a specific). For uncomplicated cases of cutaneous anthrax Murphy and his associates suggest a total of 200,000 to 400,000 units or more given at the rate of 100,000 units daily.

ARGYRIA

Argyria is the name applied to a slate or bluish-gray discoloration of the skin and mucous membranes.

Varieties and Clinical Features. Ar-

gyria may be generalized or localized. Generalized argyria follows the prolonged use of silver preparations of various kinds, especially nitrate of silver.

of neck, upper parts of chest and back, pubic or inguinal regions, and dorsa of hands and feet.

Impetigo and other superficial skin infections often complicate an atopic dermatitis.

Diagnosis The diagnosis as well as the differential diagnosis of atopic dermatitis is usually easy. It is often mis-

reaction to the patch test, then only can a diagnosis of atopic dermatitis be sustained.

Prognosis The infantile variety of atopic dermatitis usually disappears at the age of two years. The adolescent form, when properly treated results in a cure although recurrences are not uncommon. The adult form of atopic



Fig. 27 Allergic Dermatitis.

taken for seborrheic dermatitis. Seborrheic dermatitis is characterized by a diffuse sharply circumscribed erythematous and superficial scaling dermatitis. Scales are usually greasy. Itching is slight or entirely absent, and the sites of predilection are the scalp, forehead, angles of nose, postauricular and inside the ear, over the sternum, back, axillae, and pubic region.

When the family and personal history are positive for atopic disease and there is a positive immediate urticarial reaction to scratch test and a negative

dermatitis is often very intractable and presents the gravest form of dermatosis.

Prophylaxis If a family history of asthma, hay fever, atopic dermatitis, hives, or idiosyncrasy to drugs or foods is obtainable, measures should be taken to protect the infant against those contacts and foods which are known from the history to have caused this condition in the forebears.

Treatment Atopic dermatitis in infants requires specific management. The temperature of the baby's room should be about 68° F. Smooth cotton or linen

6 Certain intercurrent infections, such as common cold or influenza

7 Result of improper local treatment such as ointments or oils.

8 Emotional upsets, fatigue etc

Remissions in the course of atopic dermatitis usually result from changes in environment change of seasons, control of diet and proper local treatment

Disseminated patches may also be present on the trunk and extremities

In children over two years of age atopic dermatitis occurs either as prurigenous papules with excoriated and crusted tops occurring on the extensor surfaces of the extremities, or as lichenoid discrete flat topped brownish papules often occurring in plaques and lo-



Fig. 36 : Allergic Dermatitis (contact type)

Pathology The primary lesion of atopic dermatitis is an edematous, inflammatory acute or chronic process situated beneath the epidermis. The cardinal lesion of true eczema (the pathognomic interepidermal vesicle) is not a feature of atopic dermatitis except in infancy.

Symptoms The objective symptoms of atopic dermatitis vary with the age of the patient. In infants, prior to the age of two the eruption is characterized by erythema, papules, vesicles, papulovesicles, oozing and crusting. The sites of predilection are the face and hands.

cated on the flexor surfaces of the extremities. Oozing is rare in this variety.

In older persons, the lichen or frictional form of atopic dermatitis predominates. The lesions are more likely to be confluent and lichenification is usually present. The sites of predilection are the flexures, the neck, and eyelids. Another aspect of this condition is discussed under "Habitus Pruriticus" (see page 632).

Combinations of all or any one of the above mentioned types are not infrequently seen and may occur at any age. It is, however, unusual to find any one of the various types on the scalp back

of neck, upper parts of chest and back, pubic or inguinal regions, and dorsa of hands and feet.

Impetigo and other superficial skin infections often complicate an atopic dermatitis.

Diagnosis The diagnosis as well as the differential diagnosis of atopic dermatitis is usually easy. It is often mis-

reaction to the patch test then only can a diagnosis of atopic dermatitis be sustained.

Prognosis The infantile variety of atopic dermatitis usually disappears at the age of two years. The adolescent form, when properly treated, results in a cure although recurrences are not uncommon. The adult form of atopic



Fig. 87 Allergic Dermatitis.

taken for seborrheic dermatitis. Seborrheic dermatitis is characterized by a diffuse, sharply circumscribed erythematous and superficial scaling dermatitis. Scales are usually greasy. Itching is slight, or entirely absent, and the sites of predilection are the scalp, forehead, angles of nose postauricular and inside the ear over the sternum, back, axillae, and pubic region.

When the family and personal history are positive for atopic disease and there is a positive immediate urticarial reaction to scratch test and a negative

dermatitis is often very intractable and presents the gravest form of dermatosis.

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Treatment Atopic dermatitis in infants requires specific management. The temperature of the baby's room should be about 68° F. Smooth cotton or linen

II Certain intercurrent infections such as common cold or influenza

7 Result of improper local treatment such as ointments or oils

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Combinations of all or any one of the above-mentioned types are not infrequently seen and may occur at any age. It is, however, unusual to find any one of the various types on the scalp back

they should, therefore, be prescribed in higher concentration.

Treatment of Atopic Dermatoses in Childhood Adolescence and Adult Life
It is recognized that the younger the child the more exudative the eruption and the greater resemblance to the infantile form. In these cases the treatment is quite similar to that used in infantile eczema. On the other hand, the older the patient, the greater the tendency to a less exudative eruption and an eruption that is more infiltrated, thickened, dry and pigmented. Many of the older individuals with atopic dermatitis cannot tolerate the effects of soap, of trauma, particularly scratching and rubbing, of rapid changes of temperature and barometric pressure.

Pastes and powdery lotions applied to the trunk and extremities are preferred and are superior to ointments. To these chiefly such therapeutic ingredients as a 1 to 2 per cent resorcin, liquor carbonis detergens in 2 to 15 per cent concentration, and coal tar in 1 to 5 per cent concentration may be added. The prevention of scratching and itching is accomplished by the addition of antipruritics such as benzocaine, phenol, menthol, chloral hydrate, etc.

The skin should be cleansed either with mineral oil, 1 per cent aqueous solution of aluminum acetate or a solution of boric acid. Sedation should be limited to the use of such drugs as aspirin, bromides, and the barbiturates.

The usefulness of skin tests especially in the adult form, is problematical. Present methods of specific desensitization are also valueless. It is better to determine the principal causal or contributory allergens by means of elimination diets. In the younger group there is a higher incidence of sensitivity to food allergens while in older individuals

and adults there is a higher incidence to inhalants and other environmental allergens. It is also worth while to remember that in infants and children the tendency to multiple sensitivities is less pronounced and skin tests are more reliable.

Neurovascular instability or vasomotor neurosis should be studied. Psychotherapy endocrinologic therapy change of environment and real cures are occasionally employed with great benefit. Further aspects of therapy are discussed under *Maintaining Prognosis* (see page 832).

Sulzberger and Wolf recommend the following prescriptions which have also been found efficacious in the authors' practice:

I	
Menthol	0.25 per cent
Phenol	0.25 per cent
Liq. carbonis detergens	5.0 to 10.0
Resorcin	4.0
Benzocaine	10.0
Liq. Burron (Gifford)	30.0
Zinc oxide past	
Talcum, pudr	
Oil emul. ac	44.0
Liq. calcei, q	210.0
Use: Rub on affected parts of trunk or extremities three times each day	

II	
Menthol	0.25 per cent
Phenol	0.25 per cent
Liq. carbonis detergens	5.0 to 10.0
Resorcin	4.0
Benzocaine	10.0
Calamine Lotion, q. v.	210.0
Use: Apply three times each day	

III	
Menthol	0.25 per cent
Fat acid tannic	1.0 to 3.0
Benzocaine	3.0
Petrolatum, q. a.	20.0
Use: Apply by massage at bedtime.	

Small doses of thyroid extract, even in cases of normal thyroid function, is sometimes beneficial. This is of definite

rather than woolen or other rough materials should be used next to the skin and the outer clothing should be light and soft. The baby's skin must be kept clean and soft. Soap must not be used for cleansing. The child should be sprayed in tepid water to which bran starch or oatmeal has been added. *Inunctions* of the baby's entire skin with olive oil or mineral oil, followed by the liberal use of unscented finely powdered talcum or zinc stearate are advised.

The elimination of possible allergens is of primary importance in the diet. In children under two years of age the approach is usually very successful. The history of the effects of certain foods on the child is more valuable than the observations of skin tests. In order of their frequency cows' milk, wheat, eggs, citrus fruits, spinach, peas, tomatoes, fish and fish products are the usual dietary allergens producing atopic dermatitis.

One of the most striking facts about infantile eczema is the potent effect of environment. A great number of infantile eczemas improve and occasionally clear up entirely without therapy on being hospitalized. The child should be protected from house dust, feather pillows, mattresses, wool or silk bedding, rugs, drapes, and dyed goods to which contact might be made. Allergen proof covers will afford the necessary protection from these environmental allergens.

The Scalp. In infantile eczema the face and scalp are usually the most common sites affected. Crusts are removed from the scalp by *massaging* the scalp several times daily with 1 per cent salicylic acid in liquid petrolatum. Before beginning medication to the scalp it should be washed with soap and warm water and at least once a week thereafter. One of the most satisfactory treatments for use on the scalp as well as the

face is 3 per cent crude coal tar in borne acid ointment. This ointment should be massaged in twice daily and finally applied thickly and kept in place with a bandage. It is removed daily with mineral oil before making another application.

The Face. Impetiginized areas occurring in facial eczema are first treated by applying *compresses* of a borne acid solution or Thiersch's solution (acid borne, 12 acid salicylic & aqueous q.s. 1000). Crusting and impetiginization usually respond to this treatment in a few days and the eczematous areas may then be treated either by painting them with a thick layer of undiluted coal tar and powdered talcum or by applying a 3 per cent ointment of coal tar in zinc oxide paste. The application of crude coal tar as a paint is perhaps less irritating than any other active remedy. The tar should be left on the face for two days, then a face mask with borated petrolatum should be applied for twenty-four hours. When removed much of the tar scales, and crusts come with it. The tar painting is then repeated and after several days it is removed in like manner.

Face masks are made of soft unstarched linen or cotton cloth. In many eruptions of infancy and childhood occurring on the trunk and extremities, application of lotions, liniments or medicated vanishing creams is more effective than the use of salves. The lotions and liniments should be painted on thickly and evenly with a flat varnish brush three or four times each day. The successive coats are applied over the adhering lotion or liniment when there is much oozing. Medicated vanishing creams are equally effective.

In still other cases, *pastes* may be better tolerated. The therapeutic agents in pastes are rather slowly absorbed and

they should, therefore, be prescribed in higher concentration.

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Pastes and powdery lotions applied to the trunk and extremities are preferred and are superior to ointments. To these vehicles such therapeutic ingredients as a 1 to 2 per cent resorcin, liquor carbonis detergens in 2 to 15 per cent concentration, and coal tar in 1 to 8 per cent concentration may be added. The prevention of scratching and itching is accomplished by the addition of anti-pruritics such as benzocaine, phenol, menthol, chloral hydrate, etc.

The skin should be cleansed either with mineral oil 1 per cent aqueous solution of aluminum acetate or a solution of boric acid. Sedation should be limited to the use of such drugs as aspirin, bromides, and the barbiturates.

The usefulness of *skin tests* especially in the adult form, is problematical. Present methods of specific desensitization are also valueless. It is better to determine the principal causal or contributory allergens by means of elimination diets. In the younger group there is a higher incidence of sensitivity to food allergens while in older individuals

and adults there is a higher incidence to inhalants and other environmental allergens. It is also worth while to remember that in infants and children the tendency to multiple sensitivities is less pronounced and skin tests are more reliable.

Neurovascular instability or vasomotor neurosis should be studied. Psychotherapy, endocrinologic therapy, change of environment and rest cures are occasionally employed with great benefit. Further aspect of therapy are discussed under *Habitus Pruriticus* (see page 652).

Sulzberger and Vioff recommend the following prescriptions which have also been found efficacious in the authors practice.

I	
Menthol	0.25 per cent
Phenol	0.25 per cent
Liq. carbonis detergens	50 to 100
Resorcin	4%
Benzocaine	100
Liq. Barrois (After)	200
Zinc oxide, pol.	
Talcum, pol.	
Chloroform	400
Liq. calch. q. s.	2500

Use. Paste on affected part of trunk or extremities three times each day.

II	
Menthol	0.25 per cent
Phenol	0.25 per cent
Liq. carbonis detergens	50 to 100
Resorcin	4%
Benzocaine	100
Calamine Lament, q.	2500
Use. Apply three times each day.	

III	
Menthol	0.25 per cent
Poly acid tannin	10 to 20
Benzocaine	50
Petrolatum, q. s.	500
Use. Apply by massage at bedtime.	

Small doses of thyroid extract even in cases of normal thyroid function, is sometimes beneficial. This is of definite

value where the skin is dry and keratotic or if follicular keratosis is present. Adrenal cortex is occasionally of great value. Many cases of atopic dermatitis which cannot be relieved by any other

procedure may yield promptly to a few superficial treatments with roentgen rays.

The systemic management of cases of atopic dermatitis is based on rational general medical remedies.

ATROPHIA CUTIS IDIOPATHICA

SYNONYMS: *Atrophy of the skin, atrophoderma, dermatitis atrophicans.*

Atrophia cutis idiopathica is the direct result of changes in the normal skin which consist of connective tissue proliferation and destruction of minute elastic fibers.

Many authors classify all cases of *atrophia cutis idiopathica* as *acrodermatitis chronica atrophicans*; however, diffuse idiopathic atrophy of the skin is a clinical entity.

Varieties. *Atrophia cutis idiopathica* is either of primary occurrence or secondary, resulting from injury or nutritional disturbance.

Primary Macular Atrophy. This occurs in two forms: (1) *anetoderma erythematodes* of Jadassohn, otherwise known as *atrophia cutis idiopathica maculosa* or *dermatitis maculosa atrophicans*, and (2) *anetoderma* of Schwenninger and Buzzi.

Atrophia cutis idiopathica maculosa begins as one or more small round oval or streaklike patches. The lesion is erythematous and slightly edematous and after a short period of time the affected area becomes atrophic. Individual lesions vary from a few to 10 or more mm in diameter. The lesion is slightly depressed and on palpation shows lessened resistance. Later the surface either becomes shiny white, and simulates scar tissue, or the central part of the patch may protrude baglike and is red or yellow in color. Herniation is now demonstrable on palpation. This clinical phenomenon is common to all types of

macular atrophy and is the direct result of the loss of elastic tissue. In a given case, both new and old lesions may be present at the same time. The histological changes are the same as in acro-



Fig. 38: Postsyphilitic Macular Atrophy. Of the thigh. Many of the lesions have the appearance of trophic striae until they are spread apart. These lesions appear months after a secondary papular syphilid has disappeared. Dermic destruction has accompanied its disappearance.

dermatitis chronica atrophicans except that the changes in the vessels are the first to appear. The sites of predilection are the trunk, shoulders, and face. Subjective symptoms are absent. Regeneration with normal appearing skin after a

year or more is occasionally observed

The *anetoderma* of Schueninger and Buzz (multiple benign tumorlike new growth) is characterized by the appearance of nummular bluish-white or slate colored bladderlike lesions occurring beneath the skin. Telangiectasia may be present. Pressure with the finger over

A good formula is

Cod liver oil	27.8
Petrolatum.	30.0
Lanolin	3.0
Essential oil q. s.	

Secondary Macular Atrophy This is often a sequelae of secondary cutaneous syphilis, lupus erythematosus, tubercu-



Fig. 89 *Postsyphilitic Macular Atrophy* This is macular atrophy developing after the papules of secondary syphilis have disappeared. The atrophy largely involves the dermis; each visible lesion is soft and depressible. The epidermis has wrinkled appearance.

any one of the lesions reveals keratinization, the tumor reappearing when the pressure is released.

The disease is progressive. It is usually seen between the ages of fifty and seventy. It is more common in women. The cause is not known. Treatment is limited to the use of emollient ointments such as cod liver ointment or lipoid creams.

lous, lichen planus, and leprosy. The atrophic spots occur on the identical areas of the primary disease. Macular atrophy is also associated with *acrodermatitis atrophicans chronica*.

Treatment Vitamin A in doses of 50,000 units daily is of value. Emollient preparations applied to the skin and the avoidance of soap and water are definitely indicated.

ATROPHIA SENILIS

SYNOYMS Senile atrophy of the skin, *atrophia senilis*, *atrophia cutis senilis*, *atrophia senilis idiopathica*.

Atrophia senilis is a cutaneous degeneration in the aged which is characterized by a thinning, loss of elasticity, harshness, and a brownish or yellowish discoloration of the skin.

Varieties Two varieties of senile cutaneous changes are noted. first, the

so-called simple senile atrophy which usually occurs only on covered parts of the body and second a degenerative senile atrophy which is usually present on exposed surfaces and is referred to as *seamans* or *farmers* type of senile degeneration.

Incidence Senile changes are rarely observed in individuals under fifty; however those individuals who are thin and perspire comparatively little develop senile atrophy earlier and to a greater degree.

Etiology Atrophy of the skin may follow prolonged illness, after which it becomes smooth dry and shiny occasionally with branny desquamation and itching. The hair and nails also may be shed.

Individuals who give a history of easily becoming sunburned and rarely tan but more often develop freckles, are more likely to develop senile atrophy at an early age.

Long continued and repeated exposure to the elements and especially ultraviolet light is a causative factor as commonly seen in mariners and farmers.

Neuritis, paralysis, and various nerve diseases cause atrophy of the skin and occasionally of the underlying structure. Atrophy may also be a sequela of lupus erythematosus, scleroderma, syphilis, radiodermatitis, epithelioma, and leprosy.

Pathology In simple senile atrophy the epidermis is shrunken to a few rows of cells and the papillary borders have disappeared. The union of cutis with epithelium takes the form of a straight or slightly wavy line. The elastic tissue in that part of the cutis formerly occupied by the papillary bodies is absent and in this same region the collagenous tissue transformed into a homogenous mass still retains its power to take a stain fairly well. In the deeper layers of the cutis the degeneration of the elastic fibers has not progressed so far and although they appear rarefied they are still discernible. The collagenous tissue is also involved the fibrous structure is atrophic. This atrophy of the con-

nective tissue accounts for most of the thinning of the cutis. The staining properties of the collagenous fibers are about normal.

In the so-called farmer's type of senile degeneration of the skin involving the face back of the hands and extensor surfaces of the forearms, all elements of the skin take part in the atrophic process; however the supportive tissues are most involved. Krile believes this extensive degeneration of the supporting tissues is not necessarily due to aging of the skin but perhaps due to some chemical substance carried in the blood stream and produced in certain individuals even as early as the age of thirty-five to forty years by the metabolic processes that take place in such persons.

Symptoms The common symptom is itching. Eczematization of the skin is also observed. The skin is usually of yellowish brown color wrinkled and unelastic and occasionally desquamative. Hairs usually fall out in the involved areas. A characteristic hang of the skin on the nape of the neck (cutis rhomboidalis nuchae) with colloid degeneration is commonly seen. In this condition the brownish red to brownish-gray skin is divided into rhomboidal areas by furrows of varying depths. It feels coarse uneven and thickened. (See also "Senile Elastosis," page 643.)

Diagnosis The clinical picture and microscopic appearance of sections of the skin are pathognomonic and are unmistakable.

Complications Hypertrophy of the sebaceous glands may lead to lesions simulating adenoma sebaceum. Local accumulations of sebum and epidermal cells may form wartlike excrescences varying in size from a pea to a dime; these are referred to as seborrheic keratosis. Epithelioma may follow irritation

or traumatization of seborrheic keratoses. Telangiectases are frequently present.

Prognosis This disease cannot be cured.

Prophylaxis Prophylaxis consists of avoidance of direct sunlight and exposure to wind and inclement weather. Highly alkali soaps and too frequent bathing are to be avoided.

Treatment *Brae* or *starch* baths are soothing to the skin and should always be prescribed. *Cold cream* or *unguentum equisaphor* to which 2 per cent methylcyclic acid has been added, is helpful.

Emollient lotions containing mucilage of quince seed are very beneficial. *Vitamin A* in large doses (50,000 units) is the most effective form of general medication and should be continued for many months.

The following ointment is worthy of a trial

<i>Burrow's solution</i> (Liq. alom. acetatis)	50.0
<i>Ung. equisaphor</i> q.s.	50.0
Sig. Apply at bedtime	

None of these remedies are curative but they are not harmful and they do alleviate the dryness of the skin.

ATROPHODERMA NEURITICUM

SYNOPSIS Glossy skin, pruritic

Glossy skin is a trophoneurotic affection of the skin of the extremities, especially the fingers. It is characterized by the presence of deep red, mottled purplish, and pale patches which are glossy and ivorylike in appearance.

Incidence It may arise as a sequela or manifestation of such skin diseases as leprosy, scleroderma, syphilis, and radiodermatitis.

Etiology It is the direct result of injury or disease of the nerves supplying the affected areas. A neuritis following bullet wounds or direct trauma is perhaps the most common cause. Diseases of the spinal cord, gout, or so-called chronic rheumatism are also believed to be causative factors.

Pathology The pathology is the same as that of atrophy.

Symptoms This malady is generally preceded and accompanied by neuralgia which man feels itself as a burning pain of more or less severity. The subjective

symptoms may involve the entire limb as well as the affected area.

The skin of the affected area in the early stages of the disease is reddish or purplish, while later the color may change to gray or white. The cutaneous surface appears mottled, smooth, dry, thin, and glossy and often resembles chilblains.

Complications The nails at the free border are usually curved. Alopecia, fissuring, ulcerations, and cutaneous atrophy may follow this disease.

Prognosis Spontaneous recovery occurs in the majority of cases. Duration of the disease is variable; it may be weeks or years before resolution takes place.

Treatment The local treatment consist of applying soothing oils such as those made of quince seed, or a 2 per cent solution of menthol in cottonseed oil.

The involved parts must be protected from injury as well as extremes of heat and cold.

BALANITIS

SYNONYM: *Balanoposthitis acuta*.

Balanitis is an inflammation of the glans penis and the inner surface of the foreskin.

Varieties There are several forms of balanitis which differ in appearance as well as in their etiological background. They are referred to as (1) balanitis simplex; (2) chronic balanitis; (3) balanitis erosiva circinata; (4) balanitis erosiva ulcerosa (balanitis gangrenosa); and (5) balanitis xerotica obliterans, or kraurosis of the penis.

Incidence Balanitis is extremely rare in the uncircumcised individual. The narrower the foreskin, the greater is the likelihood of balanitis.

Etiology Seborrhea, excessive smegma, masturbation and trauma are the usual causes of balanitis. The fusospirillary organism of Vincent may be present. Acute gonorrhea, chancroid, syphilis or cancer also may cause balanitis if the glans penis is involved.

Pathology The pathology is characterized by the usual signs of inflammation such as erythema and edema to which is added a purulent and foul smelling discharge from the preputial sac.

Symptoms *Balanitis simplex* is characterized by a swollen, red prepuce and a discharge of pus from the prepuce.

Chronic balanitis may be the result of diabetes, the growth of fungi (*Oidium albicans*), prophylactic treatment for venereal disease, erythema multiforme, herpes simplex and zoster, pemphigus vulgaris, syphilis, and scabies. It may also be a symptom of a drug eruption especially antipyrine and other coal tar products. The symptoms are similar to balanitis simplex.

Balanitis erosiva circinata is a form of infectious balanitis transmitted by sexual intercourse. It is characterized by the presence of deep-seated moist lesions with loss of epidermis. These superficial lesions have a vivid color and a narrow whitish border. The painless erosions slowly spread eccentrically and may



Fig. 40 Erosive Balanoposthitis. Of two weeks duration, showing a merous Vincent organisms in dark-field examination of the secretion.

form large annular lesions with well-defined gray slightly elevated borders. A profuse, thin purulent discharge is present. The inguinal lymph nodes are enlarged. Subjective symptoms are slight or entirely absent.

It persists four to six weeks and tends to recur. Excessive preputial tissue predisposes to the condition. It must be differentiated from mucous patches and a simple balanitis, which is neither inoculable nor contagious.

Balanitis erosiva ulcerosa and *balanitis gangrenosa* are characterized by extensive ulceration, phagedena and occasionally by gangrene. The onset is quite acute and is associated with fever and prostration. The subjective symptoms are pain and tenderness. This condition may cause destruction of the penis, general septicemia and death. *Balanitis gangrenosa* may also be a complication of syphilis and chancroidal infection. In middle-aged or elderly persons, the presence of *balanitis erosiva ulcerosa* should arouse suspicion of carcinoma of the coronal sulcus.

Balanitis xerotica obliterans (*kraurosis of the penis*) is a disease of the male genitals. The cause is unknown. It is characterized by a chronic, progressive, atrophic, sclerosing process of the glans and prepuce leading to urethral stenosis. It occurs at any age, and its onset is insidious as a rule. It often follows several months after circumcision. The prepuce is replaced by a sclerotic constricting band, the glans presents diffuse or mottled patches and fissures, erosions, and even bullae accompany the electrical changes. There is gradual narrowing of the urethral orifice with almost complete stricture. In some patients, papules of lichen sclerosus and atrophicus have been found on other part of the body. A relationship between this disease, lichen sclerosus and atrophicus, and even Dupuytren's contracture has been suggested.

Prognosis. Mild cases of *balanitis* usually subside in a few days. The

chronic types usually yield to treatment after a period of weeks or months. *Balanitis gangrenosa* may end fatally.

Prophylaxis. The prophylaxis of all forms of *balanitis* consist of either circumcision or stretching of the foreskin.

Treatment. Simple *balanitis* usually responds promptly to the local application of a solution of warm potassium permanganate 1:800; solution of argentic nitrate 1:800; or *lotio niger* (black wash). Following these applications, the affected parts should be dusted with equal parts of tannic acid and zinc oxide or

Poly zinciform	1.0
Poly zinc oxide	
Pot. tale pers. sa	4.0

If the secretion is very profuse, one or two thicknesses of acroform or iodoform gauze should be placed between the foreskin and glans penis and renewed several times each day.

Balanitis erosiva should be treated by painting the affected parts with 2 per cent aqueous solution of argentic nitrate or 10 per cent protargol. Frequent applications of a suspension of sulfathiazole or of wet compresses of a solution of penicillin (1000 units per cc.) have been found beneficial. The application of the following ointment is also beneficial:

Argyrol, solution of	2.0
Balsam of Peru	1.0
Ung. zinc oxide q.s.	M. S.

The use of radiation with the air-cooled quartz light or roentgen ray has also been favorably reported.

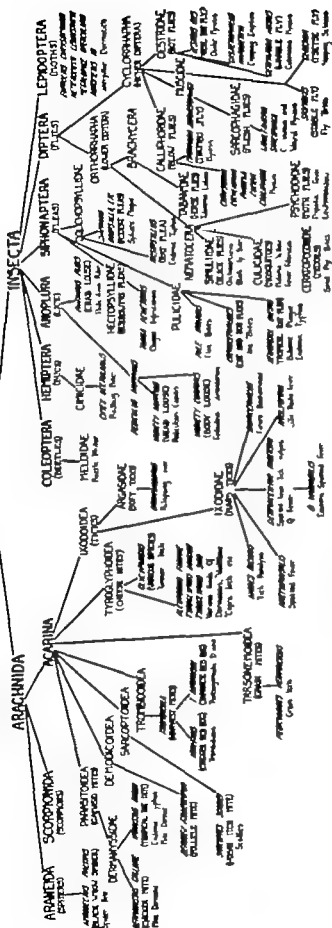
BITES AND STINGS

Introduction. Mosquitoes, spiders, flies, bees, bedbugs, gnats, blackfly, ticks, and the pediculosis family are the common biting insects seen in this country while of the stinging insects, bees, wasps,

hornets, centipedes, and ants are the most frequent.

Toxic substances, zootoxins, filterable viruses, bacteria, rickettsia bodies, pathogenic protozoa, and even helminths

ARTHROPODA



HELMINTHES (VERMES)

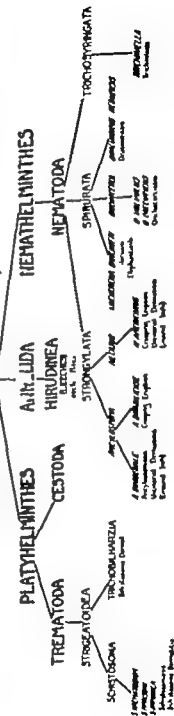


Fig. 41 Diseases due to animal organisms (ex. including protozoans) (After Oppenheimer.)

may be transmitted by bites and stings.

The itching, reddening, whealing and swelling after the bite of the pediculus, bedbug, mosquito, or spider are followed by the injection of an irritant. Contact with certain insects (minute spicules or brown patches of the browntail moth caterpillar and vesicant beetles) causes urticarial lesions, large blebs, and even an actual dermatitis. Blister beetles belong to the family Meloidae (cantharidae) and application of the pulverized body or even simple contact with many species, produces blistering of the skin. More than 800 species of blister beetles have been found in the United States. The Spanish fly (*Lytta vesicatoria*) is abundant in southern Europe and its collection to produce cantharidin requires precaution because of its vesicating properties. In any case, the lesions produced are due both to direct skin irritating materials and/or to an acquired specific sensitivity as appears to be the case in mosquito and spider bites. In chronic vagabondism (*Pediculus vestiment*) and with repeated mosquito bites, one develops an acquired immunity to the effects of the bites.

The discovery that D.D.T. is an effective insecticide against mosquitoes, flies, lice, and other arthropods has made possible the control of these transmitters of such diseases as malaria, typhus, relapsing fever and dengue.

Gnats and Mosquitoes (Culicidae)

The bites of the mosquito produce variable effects; usually the lesions resulting therefrom are urticarial or erythematous; less often bullae and rarely cellulitis or lymphangitis may occur. These insects usually inject an irritating fluid under the skin prior to sucking the host's blood. (Only the female mosquito sucks blood.) Mosquitoes transmit dis-

ease by inoculating virulent micro-organisms or by being carriers of such diseases as malaria, yellow fever, dengue and filaria.

Prophylaxis Prophylaxis consists of proper screening, the wearing of high shoes to prevent ankle bites, and the wearing of head nets and long drawers. The repellents, such as the essential oils of citronella, cassia, or eucalyptus are only of temporary value. The application of an ointment at bedtime consisting of

Opht. camphor	
Oil of eucalyptus, ss	8.0
Oil of citronella	15.0
Paraffin	90.0

usually keeps the mosquito away during sleeping hours.

Adult mosquitoes may be destroyed by spraying the room with pyrethrum or by fumigations with sulfur. The breeding places should be drained, screened, and water plants removed or their surface covered with oil.

Treatment Itching is controlled by the application of an aqueous solution of carbolic acid 1 to 30 by a 5 per cent alcoholic solution of menthol, or by a solution of weak ammonia. Desensitization with mosquito extract has been used with success.

Black Flies (Genus Simulium)

The bite of this fly is painless and is characterized by a slight hemorrhage and purpuric spot. After the course of a few hours the area itches intensely and becomes erythematopapular or nodular. They may also become vesicular and crusted, the crusts finally drop off leaving a white scar. Pain and stiffness of the involved area with regional adenopathy is often present.

The objective symptoms are controlled by the same medicaments used for mos-

quito bites. The flies are kept away by using an ointment consisting of

Oil of eucalyptus	0.25
Oil of anise seed	0.25
Oil of turpentine	0.15
Ung. aquaphor	30.00

Spiders (Arachnidism)

The true spiders (Arachnida) have poison glands and inject venom into their

imported fruit produces, when it bites, pain and swelling of the affected part for several hours. The large tarantula found in southern and southwestern United States rarely attacks man and produces only a local transient reaction when it does bite.

Spider venoms have a predominantly neurotoxic action and experimental bites or injections of the venom kill guinea



Fig. 42: Left: Spider = Caterpillar Dermatitis. Right: Spider Dermatitis. Dn is bite

prey. Many species are not particularly dangerous, but the bites of some are serious. *Latrodectus mactans* (black widow) bites are very serious. The so-called "tarantulas" of the tropics (*Solifugae*) are loathsome spiderlike creatures, but they contain no poison glands, and their bite, though severe, is not dangerous unless sepsis ensues. The "black tarantula" of Panama is one of the most venomous in this latter group. The banana spider sometimes found in

pigs with the tetanoid spasms of the muscles and bronchial contractions as in snake bite it is believed that the venom acts on the neuromuscular junctions. Some venoms, in addition contain a hemolyxin.

Important species of the genus *Latrodectus* are the red backed spider of Australia and New Zealand (*L. hasselti*) and the black widow of North America (*L. mactans*). Both spin their webs across the seats of the dry earth closets

used in country districts, and man is generally bitten on the genitals during defecation.

The venomous, female, black widow spider (*Lactrodectus mactans*) also called poison lady "black widow" shoe-button, "hourglass," "T dot," and cherry spider is the only poisonous spider in the United States. It is found in all parts of the country especially in the southern states, and in southern Canada. It is coal black with

Muscular spasms and cramplike pain are the outstanding clinical features. Pain may be so instantly severe that the patient cries out when bitten, or it may be delayed for an hour or more; it is more frequently intense radiating to the limbs, testicles or abdomen, the walls of which may be held so rigid that perforation is suspected. Features of collapse with pallor, vomiting, sweating, low blood pressure and weakness appear early. Dyspnea with respiratory



Fig. 43 Spider Dermatitis. From bite

orange or scarlet marks on the abdomen. The male is about 6 mm. long and the female about 12 mm. The abdomen is globose and has an hourglass-shaped reddish spot on its midventer. This variety of spider spins a coarse, tough web on old lumber porches, garages, and ramps, and under old floors and stones. Persons who camp outdoors may be bitten at night by spiders that have crawled into sleeping bags or bedding.

Symptoms: Locally there may be congestive edema and purpura, but often there is little obvious change. Internally, petechial hemorrhages, visceral congestion, pulmonary edema and renal changes associated with hemoglobinuria may be found.

Stridor is characteristic; the pupils are contracted and later may not react to light, and the reflexes are sluggish. Dysphagia and priapism have also been described. Dilatation of the right heart, cyanosis, pulmonary edema, and coma may occur. The temperature is subnormal at first, due to collapse but later rises, and if secondary infection ensues, the fever may persist for many days. While the symptoms may be violent and fatal, actual fatalities are few. Hematuria, characterized by the presence of oxyhemoglobin and methemoglobin in the urine results from bites of some species, including the cross spider *Epeira diadem*, and *Glyptocentrus gasteracanthoides*. Paresthesia, such as numbness

and tingling muscle spasm and weakness, may be present for months after the disappearance of the acute symptoms.

Acute symptoms generally subside after a few days but pain in the limbs and sleeplessness may persist longer. The main complication is secondary bacterial infection; this is especially a feature of *Latrodectus* bites, following which tetanus, anthrax, gas gangrene, erysipelas, cellulitis, and septicemia have all been recorded. The prognosis in most cases is good but with *Latrodectus* and certain species of *Atrax* the death rate reaches 8 per cent or higher.

Koppe-spider is the colloquial name in South Africa for at least three poisonous species of *Latrodectus*: *L. geometricus*, *L. concinnus*, and *L. indistinctus*, which cause severe symptoms and sometimes death. The two former species live in outhouses and in the walls or crevices of doors and windows, the latter in cornfields and on the veldt. In the island of Cebu in the Philippines, *L. agoyangyang* closely allied to *L. hasselti*, sometimes kills adults.

Several species of *Atrax* are found in Australia, the most deadly being the "funnel web" spider *Atrax robustus*. Local pain and profound shock result from their bite and neurotoxic features, including analgesia, respiratory stridor, contracted pupils and loss of the light reflex, follow. Death from pulmonary edema and heart failure frequently occurs.

Glyptocranium gasteracanthoides is the pruning spider of Peru so called because it is found on the underside of vine leaves in vineyards and under fallen leaves. It gives rise to much more severe local lesions than *Latrodectus*, sometimes producing gangrene, and in addition neurotoxic symptoms and hematuria, death may result.

Prophylaxis. Inspection of dry-earth

closets before use would do much to eliminate *Latrodectus* bites.

Treatment. When pain occurs immediately and the spider is seen the diagnosis is not in doubt but when there is a latent interval and local lesions are not evident as is sometimes the case in *Latrodectus* bites, cardiac failure, food poisoning or even intestinal perforation may be suspected—the latter owing to the extreme rigidity of the abdominal muscles.

If the bite is located on the extremities, immediate ligature, incision and mechanical suction are indicated as in snake bite. Locally, ichthyol and iodine are useful applications. Injections of morphine and atropine are valuable in alleviating pain and allaying laryngeal and muscular spasms. The intramuscular and intravenous injections of calcium gluconate (10 cc. of 10 per cent solution) and slow cautious intravenous injections of 25 cc. of a 10 per cent or 10 cc. of a 25 per cent solution of magnesium sulfate have recently been found remarkably effective for this purpose. The dose is repeated until there is disappearance of the toxic symptoms. Circulatory failure should be combated by saline (0.9 per cent) and glucose (5 per cent) solutions given intravenously and by injections of epinephrine solution or posterior pituitary extract and possibly coramine. Threatened pulmonary edema calls for immediate venesection. Injections of convalescent serum are worth a trial if available especially in the case of children. A horse serum *Latrodectus* antivenin, 2.5 cc. intramuscularly appears to be effective when given an hour or two after the bite.

Scorpions

Scorpions (Scorpionida) are about 8 inches long and have a partially seg-

mented body with powerful pincerlike pedipalps and paired poison glands located in the postanal segment of the spined tail, which is swung forward and plunged into its prey while simultaneously the contents of the poison sac are injected. In the tropics and subtropics scorpions of the genus *Buthus* and a number of others, such as *Euscorpius italicus* and *Centruroides suffusus*, are much feared. The sting of scorpions of the United States produces very minor symptoms; however the sting of *Centruroides suffusus*, which is found in the southern part of the United States and Mexico, is dangerous and may produce respiratory paralysis and death in children.

Symptoms. The sting is very painful and toxic symptoms soon follow; they include vomiting, diarrhea, sweating, cramps, fever and neurological features, such as trismus, stiffness of the neck, muscular paralysis, coma, and respiratory failure, may ensue. In children, death not infrequently results, and, if they survive septic infection may supervene.

Treatment. Local and general treatment as outlined for spider bite should be instituted; the application of strong solution of cocaine or the local injection of cocaine and epinephrine may be necessary to relieve the pain. A specific scorpion antivenom is available; it has been widely used in Upper Egypt and has markedly reduced the mortality among children, who are given 5 cc intravenously.

Myriapoda

The only members of interest in this class are the centipede (Chilopoda) which possesses a pair of legs to each segment of the body; the first pair are modified to form poison claws, and are connected with poison glands.

Symptoms. The bite of the small centipede in temperate climates is indicated by a red spot with a hemorrhagic dark center; occasionally an erythematous-like eruption and lymphadenopathy are present. Itching and pain are the subjective symptoms. The larger tropical centipede (*Scolopendra gigantea*) may cause local necrosis, lymphangitis, and general toxic symptoms, including head ache, vomiting, generalized pain and fever. Coma and even death may supervene in children.

Treatment. Treatment is the same as for scorpion bite; solution of cocaine being the most useful local application.

Myiasis (Fly)

The skin may be invaded by the larvae of bot flies and gadflies. Myiasis is the name applied to disease caused by the larvae of certain flies. The larvae of the Muscidae and Oestridae are the most frequent invaders. Wounds or ulcers invaded by fly maggots are called "traumatic dermal myiasis." Myiasis is produced by various species of Diptera (families Oestrididae, Gastrophylidae, and Cuterebridae) such as the bot fly and warble fly (*Dermatobia hominis* (Linnaeus), *Gasterophilus intestinalis* (DeGeer) and the *Hypoderma bovis* (DeGeer). These are specifically myiasis-producing and oviposit or larviposit in or near living tissue. Flesh flies (family *Melephoridae*) screw worm fly (*Minidae*) and certain other flies are also offenders. The clinical appearance depends on whether the larvae develop intracutaneously or subcutaneously. The intracutaneous variety is referred to as "creeping myiasis."

Symptoms. Creeping myiasis is characterized by the presence of a painful cutaneous nodule which moves in a continuous fashion, its course being marked

by erythematous ecchymotic lines. These lines disappear in a few days. The migratory character of the pain and the evanescent character of the red line are the pathognomic symptoms. The line is narrow slightly elevated tortuous, thread like and white or pink in color. It marks the course of the immature larva within or just beneath the horny layer of the skin. The rate of movement varies from 1 to 10 mm per day. The termination of the line is marked by the presence of a tender nodule. This nodule undergoes necrosis and the larva is discharged from its summit. The subcutaneous variety is characterized by ambulatory tumors which wander beneath the skin for several weeks or months until they become furuncular and the larva is discharged.

Treatment. Injections of chloroform kill the larvae in situ.

Apidæ (Bees and Wasps)

The sting of the bee or wasp is always painful and is characterized by edema and erythema at the site of injury which usually lasts about four hours. If however the sting is on the face, eyelids, and lips, it may result in edema and swelling extending over a considerable area.

Severe reactions may follow bee or wasp stings, these are characterized by generalized urticaria morbilliform eruptions, asthma and sudden collapse. These symptoms are due to (a) the toxic effect of the venom (b) the introduction of pollen from the plants with which the insect has been in contact, or (c) an allergic hypersensitiveness of the patient to the bee protein. If the sting enters a superficial vein the result may be fatal.

The poison consists of a mixture of formic acid and an organic base akin to cantharidin. The poison of the bee

is acid while that of the wasp is neutral or alkaline.

The poison sac and glands of the bee are usually left attached to the sting. The sting should therefore, be removed with forceps so that more poison will not be squeezed into the wound.

Prophylaxis. Bees are less likely to sting if one is dressed in white. Persons connected with an apiary have ready access to gloves, masks, smokers, and other prophylactic equipment.

Treatment. Solutions of weak ammonia or bicarbonate of soda are recommended. Hot applications are effective and give great relief.

The use of vinegar or lemon juice is effective in cases of hornet or wasp stings.

Fleas (*Pulex irritans*)

The common flea is found in all parts of the world. It attacks domestic animals (cats and dogs) and is temporarily parasitic on man. It inhabits crevices and cracks of floors and is prone to egg-laying in such places. Avers states that "chigoe," "jigger" or *Tunga penetrans* are synonymous terms for a variety of tropical flea. It is a more serious problem than the *Pulex penetrans*. It is possible that the American soldiers may have brought this flea back into the semitropical areas of the United States. According to Bruce and his co-workers, they feed by biting and the fertilized female burrows into the skin head first. The feet, especially the soles and beneath the toenails, are favorite sites of attack. They must be dissected out under aseptic precautions.

Fleas are important as they may be carriers of disease especially the rat flea (*Xenopsylla cheopis*) which is a carrier of plague bacillus.

Symptoms. The flea bite is characterized by a red spot, a slightly erythem

slow wheal or a wheal with a central hemorrhagic punctum. The lesion may occasionally be purpuric. The itching varies from slight to intense. Some individuals are practically immune while others attract fleas.

Treatment. Camphor and essential oil have a prophylactic effect in keeping the body free of fleas. Derris powder is also valuable for this purpose.

Local treatment consists of the application of carbolic lotion (1:40), phenolated calamine lotion, N.F. or weak ammonium water.

The clothing should be pressed and insect powder (rottenone or pyrethrum) should be placed in the bed and on infested furniture.

Cimex Lectularius (Bedbugs)

The bedbug is about 5 mm. long and 3 mm. broad and has a rusty-brown color. It has a broad short head which is separated from the body by a whitish collar. It has a proboscis containing four piercing stylets; it has 2 eyes, two antennae, and six legs. Bedbugs emit a disagreeable odor which arises from stink glands lying on the inner surface of the mesothorax. Both males and females attack the skin, the male is the smaller of the two. They are found in all countries and have the same habits wherever they may be; namely they hide in crevices during the day and venture forth only at night in quest of food.

Symptoms. Their bite produces a transitory wheal about the size of a fingernail. The bites often occur in a row numbering two or three which appear as wheals with central red puncta. Nocturnal pruritus is always present.

Prophylaxis. Prophylaxis consists of fumigation with sulfur or professional fumigation with hydrocyanic acid.

Treatment: Treatment consists of the

application of a 10 per cent menthol in alcohol or phenol lotion 1:40.

Chigger and Harvest Mites (Trombididae)

These are acari, reddish or scarlet in color. They are predatory as adults and are parasitic only as larvae. The larvae known as Leptus or harvest mite, live on animals whose blood they suck.

Symptoms. The almost invisible larvae may bury themselves partially or completely in the skin, but usually attach themselves to the skin by means of their mouth parts and feed in the same manner as ticks. They cause erythema, and an urticarial vesicular or pustular eruption. When several are close together they present an roseate appearance. Itching is usually intense. The sites of predilection are the extremities, genitalia, groins, and breast, and just below any tight constriction as a garter or a belt. One variety (*Leptus americanus*) has been found in the axillae and scalp as well as other parts of the body. The lesions may persist intermittently for weeks and frequently become infected from scratching.

It is found in summer in harvest fields, in grass bushes, swamps, and along river banks.

Trombicula Iritans (*Pulex Pennsylvanicus*). This is the chigger commonly found in America. It is bright red in color, oval in shape, and it measures from 0.3 to 0.5 mm. by 0.25 to 0.3 mm. It lives on decaying substances and fecal matter and is found in enormous numbers in swampy places during July and August.

The impregnated female penetrates and burrows into the skin, producing erythema, vesicles, pustules, and occasionally ulceration. The sites of predilec-

tion are the feet, the toes are favorite sites of attack, particularly alongside or just under the nails. The knees and scrotum may also be attacked. Adenitis may be present. Pruritus is always present.

Prophylaxis Persons walking through grass or underbrush should wear protective clothing. Infestation may also be prevented by dusting the skin with sulfur.

Treatment The mites (appearing as red specks at the center of the bite or pruritic papule) are best removed by the direct application of kerosene followed by a soap and water bath for thirty minutes. Clothes should be changed since active mites may remain in them.

Itching may be controlled by frequent application of

Menthol	0.3
Campbor	2.0
Sp. vini rect. (70 per cent)	100.0

and followed by a mild antiseptic ointment rubbed into the affected part, such as

Phenol	0.3
Ung. acidi borici (U.S.P.)	20.0

Weigel found a mixture of 20 per cent sulfur in vanishing cream to be effective as a prophylactic and as treatment.

Pediculosis Ventricosus

(Grain Mite Dermatitis, Acaro-dermatitis, Urticarioides, Grain Itch, Straw Itch, Mattress Itch)

This parasite (the *Heteropus* or *Pediculoides ventricosus*) infests straw and grain and is often present in straw mattresses. It is commonly seen in summer. This parasite does not burrow under the skin.

Symptoms The eruption, which is either urticarial, vesicular, pustular, variceloid or erythema multiformelike, de-

velops suddenly, is generalized, and is associated with itching which is almost intolerable and always worse at night. Secondary infection often occurs. Malaise, fever and moderate lymphadenitis may be present.

The suddenness of the outbreak, the intense itching and usually its appearance in seemingly epidemic form, the uniformity of the lesions, and the history of contact with grain or straw is diagnostic of *Pediculosis ventricosus*.

Treatment Treatment consists of the use of weak parasitocidal ointments such as 0.5 to 1 per cent phenol sulfur and balsam of Peru ointments. Taking sulfur by mouth is claimed to be a prophylactic measure. Lotions of rotenone (5 per cent) are helpful.

In cases of *Pediculosis ventricosus*, the bedding mattress, and wearing apparel should be disinfected. Parasitocidal ointments, such as sulfur and betanaphthol, are beneficial. In two or three weeks the malady usually disappears.

Gamasoidosis

SYNONYMS: *Chicken pickers dermatitis, fowl mite dermatitis.*

The bird chicken or fowl mite (*Demodex avium et gallinae*) may be the cause of an erythematourticarial and papular eruption, especially in those who handle chickens or clean the coops. The lesions are generally located on the hands and forearms, but the parasite may attack other uncovered parts (face, neck, or shoulders). According to Schwartz and Tulpan the parasites do not burrow. They nest in the sweat and sebaceous ducts, which become inflamed. The wearing of impervious sleeves and rubber gloves is preventive. Treatment is the same as for *Pediculoides ventricosus*.

BLASTOMYCOSIS

SYNOPSIS *Blastomycotic dermatitis, spherulomycosis humilis, dermatitis blastomycetica, cilliomycosis, verrucosa mycotic dermatitis.*

Blastomycosis is a chronic infectious disease involving the skin, and occasionally the lungs or other internal organs.

Varieties This disease may be divided into a cutaneous variety and a systemic variety. Castellani further classifies them as (1) cutaneous type; (2) mucocutaneous type, and (3) gluteal blastomycosis.

The *cutaneous type* is characterized by verrucose patches with minute abscesses; the *mucocutaneous type* involves not only the skin but also the oral mucosa and the pharynx. It is characterized by small papillomatous or frambesiform patches, which later may ulcerate. The *gluteal variety* is characterized by a diffuse induration with numerous orifices from which a thick purulent liquid exudes.

Incidence This disease occurs more often in male adults; however it has also been reported in women and in infants.

Etiology The cause of blastomycosis is a vegetable organism known as *Blastomyces*. It is round, has a granular central portion, and is encapsulated. The organism ranges from 7 to 20 microns in diameter. It stains faintly with the Gram Weigert method, but is seen in ordinary tissue section. The organism propagates itself by budding. No mycelium is seen in the tissue; however an abundant growth of mycelium develops on culture medium. Animal inoculation usually gives positive results and is characterized by the production of granulomatous nodules in the skin, lungs, kidneys, etc. from which it is possible to culture the organisms. The best media are Beerwort and glycerin and glucose agar. According to Ormsby the best dye for staining the organism in tissue sections

is Unna's methylene blue and orange tannin combination.

Pathology According to F. H. Montgomery the characteristic changes occur in the rete, which is the seat of extensive hyperplasia sending down processes deep into the corium. These processes are exceedingly irregular and send out branches in all directions. They contain miliary abscesses which are characteristic of this infection and often are sufficiently large to be seen by the naked eye. They contain leukocytes, nuclear fragments, epithelial debris, red-blood cells, blastomycetes, and frequently giant cells. The rete is edematous and infiltrated with polymuclear leukocytes. The corium is the seat of subacute, chronic, and occasionally acute inflammatory changes and miliary abscesses. The organisms are found in the miliary abscesses, giant cells, and occasionally in the granulation tissue. In the deeper form of blastomycosis, the tissue changes resemble those of tuberculosis except for the lesser degree of central necrosis.

Symptoms The yeastlike organisms (*Blastomyces*) produce a variety of clinical entities differing in appearance in various parts of the world.

Based on the clinical appearances of the disease as well as on the character of the causative agent, the following three types have been distinguished: (1) the North American type (Gilchrist), (2) the European type of Husse (Buschke) and (3) the South American type. This discussion is limited to the type commonly encountered in North America.

The North American type has a superficial location in the skin. The sites of predilection are the face, hands, wrists,

and forearms but no part of the body is exempt. The earliest lesion is a pin head to pea sized papule or papulopustule. This early lesion gradually enlarges either by peripheral extension or by the development of new foci until



Fig. 44: Blastomycosis.
(Courtesy of Dr. C. C. Thomas.)

after several months it has attained a diameter of from 1 to 20 cm. Crusting is present almost from the beginning and the diseased patch is elevated from $\frac{1}{4}$ to $\frac{1}{2}$ inch above the plane of the normal skin. On removal of the crust, the underlying lesion consists of red or purple, irregular papillomatous tumors bathed in seropurulent fluid. The characteristic symptom is the abruptly sloping border which is dark red or violaceous in color and studded with milium abscesses that appear as yellow points. Owing to auto-inoculation multiple lesions may occur. Ordinarily the patches extend peripherally and heal in the center with the formation of white atrophic scars not

unlike those of lupus vulgaris. The disease usually involves only the skin where it is limited to one or more circumscribed patches. Sometimes it becomes systemic.

Systemic blastomycosis often involves the lungs, liver, spleen, the bones (especially the bones comprising the elbow joint, the tibia and vertebrae), the joints, the brain, meninges, gastrointestinal tract, etc. The lymph nodes are rarely involved.

The characteristic symptom of systemic blastomycosis is the occurrence of multiple subcutaneous nodules and ab-



Fig. 45: Blastomycotic Dermatitis.
Complete resolution of lesion in about seven weeks following radiation with roentgen-rays and the internal use of Lugol's solution. (Courtesy of Dr. J. D. Bush.)

cesses, which appear in successive crops and subsequently develop into ulcers of varying shape and size. The symptoms depend upon the organs and tissues that are involved. Fever of 103 F., extreme

weakness, progressive wasting and anemia are usually present.

Diagnosis The fully developed plaque of blastomycosis presents a cauliflower-like appearance with clefts and fissures of varying depth from which a seropurulent

exudate oozes from small sinuses. This is so characteristic that errors in diagnosis should not occur.

Prognosis The prognosis of cutaneous blastomycosis is good. Recurrences are not uncommon. The prognosis of systemic blastomycosis is grave and the mortality is about 90 per cent.

Treatment Best results have followed the internal use of large doses of



Fig. 46 Chromoblastomycosis.
(Courtesy of Dr. V. Pardo Castello.)

iodides (800 to 300 grains daily) and roentgen irradiation (2500 r). Vitamin B should be given in large doses in conjunction with the above mentioned

the discovery of the budding *Blastomyces* in the pus or in sections is essential for definite diagnosis. Cultures may be necessary.

Tuberculosis verrucosa cutis sometimes closely resembles it, however the limited distribution and slow development of tuberculosis verrucosa cutis should prevent error of diagnosis. In sporotrichosis, the lesions consist of subcutaneous abscesses and involve the epidermis only secondarily. Vegetating syphilitic usually occurs on the scalp, or about mucous membranes and is accompanied by the signs of syphilis. Bromide and iodide eruption may sim-



Fig. 47 Chromoblastomycosis.
(Courtesy of Dr. Henry H. Perlman.)

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(Courtesy of Dr. C. C. Thomas.)

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CALCINOSIS

Calcinosis is deposition of lime salts in the tissue.

Varieties Two types of calcinosis are recognized: calcinosis universalis and calcinosis circumscripta.

Calcinosis universalis is characterized by the appearance of tumors in the corium, subcutaneous tissue, muscle, fascia, and about tendinous insertions of

vitamin D and parathyroid deficiency are causative factors of universal calcinosis. There is "metastatic" calcinosis (bone calcium redeposited in skin) "metabolic" (most common and a true disturbance in calcium metabolism) and local.

Pathology Microscopy reveals calcium primarily in the subcutaneous tissue but also in the dermis. The calcified



Fig. 48 *Calcinosis Universalis*. A number of calcareous deposits were present elsewhere in the skin.

joints ranging in size from a pea to a walnut.

Calcinosis circumscripta involves the upper extremities, especially the hands, lobes of ears, and scrotum. The fingers become swollen and present calcareous nodules.

Incidence No race or age is exempt.

Etiology Local disturbances of nutrition favor the deposit of calcium in the tissue. The pH of the blood, sunlight,

patches are surrounded by connective tissue. Blood calcium and phosphorus levels are normal. Sutton believes calcinosis to be closely allied to xanthoma. In some cases the blood cholesterol has been high. X-ray examination reveals subcutaneous calcified tissue deposits of calcium phosphate and carbonate.

Symptoms The integument overlying the calcified growths is red, adherent, and may present perforations. The result

therapy. Intramuscular injections of *colloidal copper* are recommended and should be used in conjunction with the large doses of potassium iodide.

The local use of 1 per cent aqueous solution of *copper sulfate* or a saturated solution of *gentian violet* or tincture of *iodine* have all been favorably reported.

In addition to the foregoing Ravaut recommends the intravenous use of *compound iodine solution* (U.S.P.) given as follows: 1.5 cc mixed with 1 gm of *sodium thiosulfate* given daily for four days; then 1.8 cc mixed with 1 gm of *sodium thiosulfate* given daily for the next six to eight weeks.

Chromoblastomycosis

This is a form of blastomycosis due to several fungi, especially the *Phialophora verrucosa* and the *Hormodendron pedrosoi*.

The disease is common in tropical and subtropical countries, but is occasionally seen in the United States.

The lesions usually occur on the lower extremities. They are single or multiple, advance slowly, persist for years, and are relatively symptomless.

According to Pardo-Castello, the lesions are of five types: verrucous, syphilitoid, tuberculoid, psoriasisiform or cicatricial, and elephantiasic. The verrucous wartlike nodules which may or may not ulcerate are the commonest form.

The diagnosis commonly rests on finding the organism in the scales or pus or in tissue sections, in which they appear as round yellow bodies within the giant cells of the granuloma. As treatment Pardo-Castello advises electrocoagulation or deep x-ray therapy. Iontophoresis with copper sulfate has been found beneficial in some cases.

in the form of a plaster or in collodion. Avoiding further pressure, by placing a felt pad on foam rubber behind the callus to raise a depressed metatarsal head, will reduce the tendency to hyperkeratotic formation. The horny mass may also be softened and removed by soaking the foot in hot water preferably preceded by the salicylic acid application. A caustic dose of roentgen rays will often remove a corn or a callus for several months. Recurrences are usual. Shielding with moleskin or felt pad behind a callus to raise a depressed metatarsal is helpful at times.

Röntgen ray treatment (600 to 1200 roentgens) is specific for painful vascularized callosities. For painful vascularized corns, roentgen ray treatment in doses of 600 roentgens at ten-day intervals to three doses is specific.

The lesion should be closely shielded.

"Corns may be dissected out after injection of 2 per cent procaine solution around it in a fan-shaped manner. The injection alone often relieves the pain. Sinuses which occasionally complicate corns should be thoroughly cauterized with phenol introduced along their lengths with a fine applicator. It may be necessary to open curet and pack such a sinus or even completely excise it. Soft corns are best treated by dissecting out as much of the softened keratotic material as possible followed by the local application of 50 per cent silver nitrate solution and raising the head of the fourth metatarsal bone with a foam rubber shield. The involved toes may be kept apart by a small felt wedge lamb's wool or piece of foam rubber (Montgomery).

ing ulcers discharge a creamy oleaginous material. Healing leads to scar tissue formation and may produce contractures. The wrists, knees, and hips are usual sites of predilection.

Diagnosis. The cutaneous deposits of calcinosis are distinguishable from cysts and the deposits of gout by roentgen ray investigation or by biopsy.

Treatment. Treatment is of no avail in calcinosis universalis, although a *keto-genic diet* has been advised. Sutton obtained favorable responses in a patient in whom he injected *parathyroid extract*. *Disodium phosphate* in doses of 1 gm. daily *syrup of iodide* and *potassium iodide* are worthy of a trial. Lesions of calcinosis circumscripta are excised.

CALLOSITIES AND CORNS

Definition. The *callus* is a chronic traumatic dermatitis with thickening of the derm and hyperkeratosis.

The corn is a traumatic hyperkeratosis with inflammation at first and later atrophy of the derm. It is a conelike formation with the apex of the cone directed downwards against sensitive subjacent structures and bony prominences, in which the epidermic cells have acquired such abnormal qualities that they persist often long after removal of the cause.

Symptoms. *Callosities* are round or oval pigmented or yellowish variably sized hornlike patches which are thick and firm on palpation. They are commonly located on the palms, soles, and toes especially at the head of the first metatarsal (in hallux valgus and often as part of a bunion) and on finger and buttocks of equestrians.

They often are occupational in origin and represent a cutaneous defense reaction the result of friction or pressure. They are not painful unless they become inflamed or vascularized. In the latter case a subepidermal abscess may occur with lymphangitis and slight fever. When a callus is pared the papillary lines are all clearly visible. There is no central core as in a corn.

The *corn* is seen only on the feet where it develops as a result of chronic

pressure of ill fitting shoes. Corns are found usually on the fifth toe and over the phalangeal joints. They are single or multiple and are termed "hard" or "soft" depending on their location. They are always painful on pressure and often sore in wet weather. Plantar warts are often mistaken for corns. Soft corns occur between the toes when they become macerated by sweat. Infection, lymphangitis, and abscess formation may occur spontaneously or as the result of improper treatment. Vascularized corns are very painful and are usually located on the first or fifth metatarsal heads (neurovascular corn of Montgomery).

Soft corns develop on the lateral surfaces of the toes. These interdigital lesions show a central depression, a whitish gray color and softness due to heat moisture and resultant maceration.

Treatment. Callosities and corns disappear completely when the cause is removed. Roomy or orthopedic footwear is the best single therapeutic item. In most instances, excessive callus on plantar areas indicates incorrect foot posture or actual deformity within the foot structure. Orthopedic correction is often indicated. Both callosities and corns are treated in addition by regularly removing the hyperkeratotic surface with a sharp scalpel or with such chemical agents as 20 to 40 per cent *salicylic acid*.

be properly referred for such management, if and when indicated

Etiology

Incidence. *General.* The number of cases of skin cancer extant in this country at any given time is probably about six to eight times the 3500 to 4000 persons who needlessly die yearly from this disease. About 2.65 per cent of all cancer deaths in the United States are attributed to skin cancer a disease which develops sometimes for years in plain sight of the victim.

Samples from 14,000 tumor cases of all types reported to the Division of Cancer Control of the State of Pennsylvania show that skin cancers comprise 13.5 per cent of the tumor samples in males and 8.1 per cent in females. This suggests that skin cancer is considerably more common than generally supposed.

Frequency of Skin Cancer in Different Sites of the Body. The site of a cutaneous cancer is one of the factors to be considered in planning treatment. An analysis of 1792 skin cancers (excluding the lip, vulva, and anus) at the Oncologic Hospital showed that 1628, or 88 per cent, involved the skin of the head and neck. The distribution of these lesions was as follows: the scalp, 29; the eyelids, 133; the ear 180; the neck, 180; the nose, 380 and the open areas of the face 639. The remaining 164 cases were distributed as follows: hand, 51; forearm, arm, and axilla 19; lower extremity including groin, 37; trunk, 81; penis and scrotum, 23. (See Table 1.)

McFarland, Ciccone, and Gelehrter believe that their study in plotting the

TABLE 1 CUTANEOUS CANCER, RELATIVE FREQUENCY IN VARIOUS SITES. AMERICAN ONCOLOGIC SERIES (1792 CASES)

Site of Lesion	No. of Cases	Ratio	Per Cent
Open areas of face	639	1.3	33
Nose	380	1.4	21
Neck	180	1.9	11
Ear	180	1.10	10
Eyelids and eyelids	133	1.11	9
Trunk	81	1.25	4.5
Hand	51	1.03	3
Lower extremity	37	1.44	2.1
Scalp	29	1.80	1.6
Penis-scrotum	23	1.73	1.3
Forearm and arm	19	1.80	1.0

Temple, forehead, cheek, chin.

site of origin of basal cell carcinomas supports but does not settle the question that basal cell carcinomas are dysontogenetic tumors which originate in imperfections in the closure of the embryonal facial fissures.

Age. Carcinoma of the skin is a disease of advanced age, as may be seen from the Census Bureau reports. No apparent age difference exists when squamous-cell cancer patients and basal-cell cancer patients are compared. Cutaneous carcinoma ranks second in cause of cancer deaths after the eightieth year from seventy to seventy nine, it ranks fifth from sixty to sixty nine, it ranks sixth, from fifty to fifty nine, it drops to eighth, and from forty to forty nine, it ranks ninth in order of cancer deaths. It is rare in the young although it may be expected to appear in xeroderma pigmentosum, always in multiple form. Over a thirty year period, Hall and Bagby at the Barnard Free Skin and Cancer Hospital, collected only 28 proved and 32 clinically diagnosed cases of skin cancer in patients under thirty years of age. In the Oncologic Hospital

Estimated from statistical data of the Division of Cancer Control of Pennsylvania.

*Cases studied were from the American Oncologic Hospital and the Philadelphia General Hospital, Philadelphia.

CANCER OF THE SKIN

S GORDON CASTIGLIANO M.D., F.A.C.S

Introduction The end results in the treatment of epithelioma of the skin indicate that the management of such lesions is not the simple and settled problem many would believe. In the hands of the therapist who has a full knowledge of the pathology and possible course of carcinoma of the skin and who has at his disposal the necessary armamentarium, there is no question that the results throughout the country are uniformly good; however cancer clinics show a high percentage of referred recurrent cases to have a history of previous inadequate treatment. This suggests that many patients are not receiving the full benefit of existing knowledge concerning the management of cancer.

At the American Oncologic Hospital approximately 1722 cases of epidermoid carcinoma of the skin were available for study up to 1938. A review of 300 unselected cases seen after 1930 showed that 84 received previous inadequate treatment in the form of irradiation or surgery; 30 received acid caustic plaster carbon dioxide snow or other forms of treatment. From this it is apparent that 120 cases or 40 per cent of the series had been primarily unsuccessfully treated before referral to the clinic. This experience is not singular as Warren reports a series from Hunting Memorial Hospital, where 23.8 per cent had received previous sublethal therapy. Driver and Cole report that 25 per cent of their series had received previous inadequate therapy.

Undoubtedly some of the 3500 to 4000 deaths which are yearly attributed to cancer of the skin may be traced to

delay in seeking treatment on the part of the patient, but the majority of deaths appear attributable to inadequate primary therapy at the hands of unskilled and insufficiently trained men.

In order to manage carcinoma of the skin properly the ideal therapist must be able to treat not only a small primary lesion but he must also be in a position to treat extensions, metastasis, and other complications. A thorough knowledge of the vagaries of the disease under treatment is essential. In addition he must have a good knowledge of radiation physics in general and a thorough knowledge of his personal equipment, which should include radium. The availability of equipment must be such as to assure satisfactory management of all phases and complications of carcinoma of the skin. If the therapist is not an oncologist capable of managing his cases surgically when surgical procedures are indicated he must at least have available a surgical consultant, experienced in cancer work. Lastly and as important a factor as any mentioned in the successful treatment of skin cancer is the maintenance of a carefully controlled follow up clinic where patients are checked regularly.

Because many patients with skin cancers gravitate to the dermatologist, it is vital that he develop a more thorough knowledge of the management of the disease. It is equally important that each dermatologist should evaluate accurately the limitations of his equipment in order that patients who would be served better by the use of facilities other than those at his disposal may

be properly referred for such management, if and when indicated

Etiology

Incidence. *General.* The number of cases of skin cancer extant in this country at any given time is probably about six to eight times the 3500 to 4000 persons who needlessly die yearly from this disease. About 2.63 per cent of all cancer deaths in the United States are attributed to skin cancer a disease which develops sometimes for years in plain sight of the victim.

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Frequency of Skin Cancer in Different Sites of the Body. The site of a cutaneous cancer is one of the factors to be considered in planning treatment. An analysis of 1722 skin cancers (excluding the lip, vulva, and anus) at the Oncologic Hospital showed that 1528, or 88 per cent, involved the skin of the head and neck. The distribution of these lesions was as follows: the scalp, 29; the eyelids, 135; the ear, 156; the neck, 180; the nose, 389 and the open areas of the face, 630. The remaining 194 cases were distributed as follows: hand, 51; fore-arm, arm, and axilla, 19; lower extremity including groin, 37; trunk, 61; penis and scrotum, 25. (See Table 1.)

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Estimated from statistical data of the Division of Cancer Control of Pennsylvania.

*Cases studied were from the American Oncologic Hospital and the Philadelphia General Hospital, Philadelphia.

series only 18 of 313 patients were under forty years of age

Sex Skin cancer is more common in men in our series, 62 per cent were males, while only 38 per cent were females. The average age in men was 64.3 years and in women 60.1 years

Climate Skin cancer is stated to be more common in the temperate zone but many cases are reported from the

be of less frequent occurrence in the Negro in the ratio of 3:1 or more.

Heredity The influence of heredity to cancer of the skin has not been studied sufficiently to permit of any conclusions. Even granting the possibility of an hereditary predisposition environmental factors dominate.

Stimulating Causes Although the mechanism which operates when orderly



Fig. 49 Squamous-Cell Carcinoma. Left Basoid epidermoid carcinoma, squamous-cell type Grade IV. Bilateral cervical and preauricular metastases. Right Lesion healed after 6000 of fractionated high voltage therapy. Metastases controlled after 1800 of high voltage roentgen-ray treatment to large, preliminary bilateral cervical fields, followed by treatment of individual nodes through small cones of a total dose of 5400. Interstitial radon in plantation employed following roentgen irradiation.

tropics. It is thought to be more common in dry windy regions.

Complexion. Blond individuals whose skins are inclined to freckle are more liable to skin carcinoma than those whose skin has the ability to tan. Corlett, in a series of cutaneous cancers found 63 per cent in fair skinned persons with dark hair while only 7 per cent were found in individuals with both dark hair and deeply pigmented skin. In temperate zones at least, it appears to

epidermal cells suddenly take on neoplastic characteristics, is unknown, some contributing or exciting causes which can set this mechanism in motion are known. These stimulating factors will be briefly outlined.

Chronic Irritation Irritation from long existing abnormal conditions of the skin, such as lupus vulgaris, discoid lupus erythematosus, varicose ulcers, acrodermatitis atrophicans, osteomyelitic fistulae may occasionally eventuate in cutaneous

carcinoma: The latency period before malignant degeneration occurs is measured in years; for example the latency period for the rare carcinoma developing on the basis of varicose ulcers averages about twenty years for osteomyelitic fistulae, nearly thirty years.

Other important precancerous lesions of the skin are the incurable xeroderma pigmentosum, the dangerous senile keratosis, vulgar leukoplakia, cutaneous horn etc.

Physical Agents. SOLAR RAYS That the rays of the sun play an important part in the production of cancer of the skin has been recognized since the days of Unna, in 1894. The relatively high incidence of cutaneous carcinoma in sailors, farmers, lumbermen, fishermen, and other outdoor workers exposed to the drying and irritating effects of sun and wind is well known. It has been alleged that carcinoma of the skin in sailors has a tendency to prevent cancer of internal organs. Warren and Gates conclude from their observations that cancer of the skin does not protect against the development of cancer in other body sites. The work of Killy and Hill arrives at the same conclusion. Experience at the Oncologic Hospital is in agreement with these observers.

X-RAYS AND RADIUM The injurious effects upon the skin and possible malignant degeneration of dermatoses which may develop from injudicious exposure to radium or the roentgen rays are too well known to discuss here. The total of reported cases collected from the world literature of occupational roentgen and radium cancers of the skin numbers only 125. This figure undoubtedly is considerably lower than the actual one. Although occupational and accidental roentgen or radium carcinoma are well known, the growing number of skin can-

cers developing following therapeutic irradiation is less well known or appreciated. A significant number of so-called therapeutic cancers appear after roentgen irradiation of a previously existing skin affection, such as lupus vulgaris and lupus erythematosus. Other skin affections reported to develop carcinoma following prolonged exposure to roentgen



Fig. 30 Squamous-Cell Carcinoma of right temporal area. Basal-Cell Carcinoma of nose, near inner canthus, in the same patient (1943)

radiation are psoriasis, eczema, pruritus ani, pruritus vulvae and trichophy tons.

It is conjectural how much greater the incidence of therapeutic cancer might be if patients suffering with advanced carcinomas of various organs could survive the disease for which two or three cycles of intense irradiation were successfully used.

THERMIC BURNS AND FROSTBITES Included with physical agents may be mentioned the development of cancer on the basis of thermic burns and frostbites. It is thought by some that cancers re-

TABLE 2 SITES OF LESIONS IN 401 CASES OF SKIN CANCER.

Type of Epidermoid Carcinoma	Nose		Temple† Cheek Chin		Eyelid*		Ear		Total of All Lesions and Per Cent	
	No.	Per Cent	No.	Per Cent	No.	Per Cent	No.	Per Cent	No.	Per Cent
Basal cell	15	77.5	185	57.0	31	55.0	15	33.0	270	67.5
Cystic basal cell	2		9		2		0		13	
Squamous cell	4	20.0	114	33.8	19	32.0	20	44.0	157	39.0
Bisquamous cell	9	15.5	31	9.2	7	12.0	4	11.0	51	12.5

Cases seen, 1930-1938

† Cases seen, 1930-1941



Fig. 53: Squamous-Cell Carcinoma. Epidermoid carcinoma of the squamous-cell type showing connection to the epidermis and invasion. Not occasional concentric arrangement of cells with central cornification suggestive of early pearl formation.

be affected part. In the presence of an ulcerated lesion, enlarged tender regional nodes are almost always due to infection; the adenopathy subsides after adequate treatment to the local lesion. In contrast to the tender inflammatory nodes, metastatic nodes ordinarily are hard and insensitive with a tendency to become fixed to surrounding tissue.



FIG. 53. Squamous-Cell Carcinoma. Cornification in typical pearl formation seen in squamous-cell carcinoma.

Histopathology The microscopic appearance of squamous-cell cancer varies widely according to its stage of development: its degree of anaplasia; the presence of infection; the effects of previous treatment, etc. (see Figs. 53 to 57). As the squamous-cell tumor develops, epithelial proliferation produces a widening of the rete pegs with eventual disruption of the basal-cell layer. The disorganization of the basal-cell layer continues and may be lost in the path of invasion of tumor cells which earlier may appear as small nodules, later as large projections, and finally as sheets of neoplastic cells. The connective-tissue stroma in the more active forms is scanty; more abundant in the slower growing less malig-

TABLE 3. BRODER'S CLASSIFICATION

Grade of Tumor	Per Cent Differentiated Cells	Per Cent Undifferentiated Cells
Grade I (least malignant)	100 to 75	0 to 25
Grade II	75 to 50	25 to 50
Grade III	50 to 25	50 to 75
Grade IV (most malignant)	25 to 0	75 to 100

nant types. If searched for prickles can be found connecting the cells. Epithelial pearls and cornification are frequently present and are ordinarily regarded as diagnostic for this type of tumor. The frequency with which mitotic figures are encountered varies with the degree of malignancy.

PACODERMATITIS-LIOMATOUS HYPERPLASIA At times, this is associated with benign ulceration and occasionally presents a histologic picture of squamous-cell carcinoma so typical that even experienced pathologists may be deceived. Various writers describe criteria by which differentiation can be made. The clinical course settles the issue if the condition is suspected and the lesion is untreated.

MICROSCOPIC GRADING Seen in the least malignant form of squamous-cell carcinoma are differentiated adult squamous cells, pearl formation, and cornification in abundance (see Figs. 53 and 54) and when grading the degree of malignancy from one to four it is graded one. The tendency toward production of the adult form of epithelium decreases and evidence of pearl formation becomes more and more meager as the grade of malignancy of the tumor increases. Little evidence of hornification or pearl formation can be seen in tumors which are of Grade III malignancy. One finds in

Grade IV tumors sheets of undifferentiated highly anaplastic cells with hyperchromatic nuclei and frequent mitoses with scant evidence of pearl formation or hornification. The grading of malig-

nant epithelial tumors was originated by Broders (see Table 3)

It should be obvious to all that the grading of one small section of a tumor may not be representative of the entire

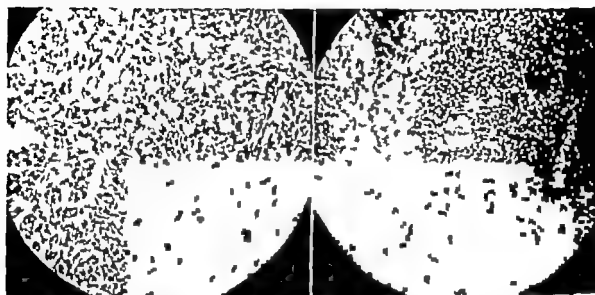


Fig. 55: Squamous-Cell Carcinoma. Left: High power view of sheet of cells in an undifferentiated squamous-cell carcinoma. Right: Low grade of malignancy

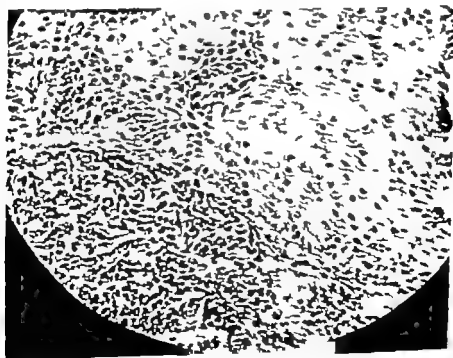


Fig. 56: Squamous-Cell Carcinoma. Note the contrast between the hyperplastic and dyskeratotic epithelium and the underlying undifferentiated cancer cells. This was a typical noncornifying squamous-cell carcinoma.



Fig. 57 Squamous-Cell Carcinoma. Recurrent squamous-cell carcinoma, showing invasive strands of neoplastic squamous cells in dense stroma. The superficial round-cell infiltration is in keeping with the clinical observation of ulceration.



Fig. 58 Basal-Cell Carcinoma. Left Of forehead. Not rolled, pearly edges and depressed, ulcerated center. Right Of forehead. Note translucent appearance.

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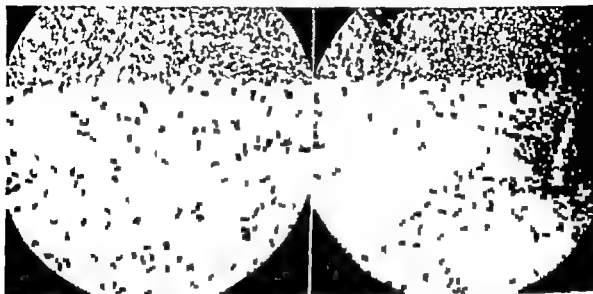


Fig. 55: Squamous-Cell Carcinoma. *Left:* High power view of sheet of cells in undifferentiated squamous-cell carcinoma. *Right:* Low grade of malignancy.

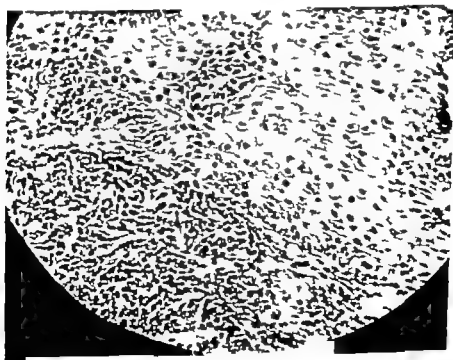


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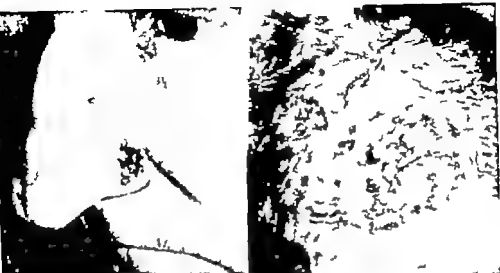


Fig. 60 Epithelioma. *Left* Of nose *Right* Of scalp. Not characteristic harder



Fig. 61 Basal-Cell Carcinoma (rodent-ulcer type) Note keratoid scum on forehead.



Fig. 62 Basal-Cell Carcinoma (rodent-ulcer type) Patient survived period of fourteen months after photograph was taken. Pain relieved by alcohol injection of gasserian ganglion.

tumor. The error however can be only in one direction. A microscopic section any representative portion of which shows a high degree of anaplasia undoubtedly represents the truer grade of the tumor. If such an area of the neoplasm is missed probably the tumor will be graded lower than its true degree of malignancy.



Fig. 391. Basal-Cell Epithelioma.

Despite the many words printed and spoken questioning the value of grading the practice still continues in most hospitals. Broders shows a remarkable degree of correlation between tumor grade and end results.

Microscopic Appearance of Metastasis. The appearance is usually that of an anaplastic squamous-cell carcinoma, although pearls and hornification are occasionally seen. Giant cells are sometimes seen and are alleged by some to be due to irradiation however they are also seen in nonirradiated nodes.

Broders found the average grade of squamous cell carcinoma to be 2.10

whereas the average grade of those which metastasize was 2.03. In our series of carcinoma of the face the average grade of lesions which metastasized was 3.2.

Microscopically the discovery of epithelial cells in a lymph node, when only a small specimen is available for study as is frequently the case in an aspiration biopsy should be conclusive evidence of metastasis.

Epidermoid Carcinoma—Basal Cell Type. This tumor was first isolated as a clinical entity by Jacob of Edinburgh, in 1827 and as a histopathological entity by Krompecker in 1900. The origin of this tumor has been ascribed to such various structures as the basal cells of the epidermis, hair follicles, and sweat glands. Glasunow, McFarland et al and others have suggested a dysontogenetic origin.

Clinical Appearance. The lesion frequently begins as a reddish, painless papule, frequently mistaken by the patient for a pimple that wouldn't come to a head. Not infrequently a history may be elicited of attempted expression of the supposed contents. After a variable period of time, ranging from several months to the more usual period of several years, the patient is struck by the fact that the tumor has increased in size. The lesion sooner or later ulcerates and tends to spread peripherally; the advancing edge is indurated. Ordinarily invasion of the deeper structures is not seen until late and then usually only after incomplete surgical removal has broken down the barriers. Categorically it may be stated that a basal-cell carcinoma does not metastasize, although relatively rare references in the literature cite examples of metastasis to lymph nodes.

Today one should be trained to recognize malignant skin lesions when they



Fig. 60 Epithelioma. Left Of nose Right Of scalp. Not characteristic border



Fig. 61 Basal-Cell Carcinoma (rodent-ulcer type) Note hernious or nodule on forehead.



Fig. 62 Basal-Cell Carcinoma (rodent-ulcer type) Patient survived period of fourteen months after photograph was taken. Pain relieved by alcohol injection of gasserian ganglion.

are 2 to 3 mm or less in diameter. Resident oncologists develop this skill after eighteen to twenty-four months of training.

The typical basal-cell tumor with a rolled pearly edge and depressed ulcerated center may be recognized by all (see Figs 58 to 60). Other clinical types exist such as the flat, morphea-like type

seen today. Death from uncontrolled basal-cell carcinoma comes slowly after the dimensions have increased so as to interfere with the nutrition of the patient. Hemorrhage sometimes is the cause of death but more frequently cachexia invites a terminal bronchopneumonia.

Histopathology The microscopic appearance is usually typical (see Fig. 63



Fig. 63: Basal Cell Carcinoma. *Left:* One of five cancers involving the skin of a patient who eventually died of carcinoma of the rectum. Typical, deeply staining nuclei in closely packed cells arranged in nests. *Right:* Involving the tragus of the ear. Various sized, deeply staining cellular masses with an orderly peripheral palisade arrangement of more basophilic cells characteristic of the basal-cell cancer. The section does not happen to show attachment to overlying atrophic skin.

the field fire type, the translucent plaque, the cystic type, the nodular type, the pigmented type, the superficial cicatrizing type, the papillary type, and the rodent ulcer type (see Fig. 61). The latter designation we reserve for the deep ulcerating flat-edged lesion which has taken on sudden increased growth, has been radiovaccinated by previous inadequate therapy, and only too often cannot be permanently controlled (see Fig. 62). Such rodent ulcers are not commonly

and 64). Sections show variable sized groups or compact masses of deeply basophilic cells. These cell masses show a well-delineated periphery of typical basal cells arranged in palisade. The entire tumor mass is usually well circumscribed and appears to be separated from the overlying epithelium.

Ordinarily peripheral invasive qualities are absent, although in more advanced and in previously inadequately treated lesions long narrow arms of



Fig. 64 Basal-Cell Carcinoma. Flat basal-cell carcinoma involving the skin of the suprapubic region, measuring 6 x 4 cm. in largest diameter clinically diagnosed as Paget disease. This microscopic view is typical of the entire slide. Note the attachment of neoplastic-cell clusters in the epidermis.



Fig. 65 Basosquamous-Cell Carcinoma. Left Recurrent mixed basosquamous-cell carcinoma of right temporal region. Treated by local electro-surgical excision and massive doses—1200 r.—of roentgen-ray treatment to each of five recurrent areas. Additional fractional high voltage irradiation to two small areas of persistent disease. Right Lesion healed. No evidence of recurrence three years later.

reaching cells can be demonstrated for some distance beyond the bulk of the tumor

The hyperchromatic cells which make up the cell nests vary in form from round to pencil or spindle-shaped cells depending upon such environmental factors as pressure etc Necrosis of variable



Fig. 66: Basosquamous-Cell Carcinoma. Epidermoid carcinoma, indeterminate or basosquamous-cell type in old skin of nose. Note small basal-cell carcinoma of right cheek.

sized groups of cells may produce cyst like spaces. Inflammatory changes are not infrequently seen in the presence of ulceration. In our laboratory basal-cell tumors which show any tendency toward the squamous-cell type, such as prickles, formation, hornification, or pearls, are classed in type as basosquamous.

Basosquamous Cell Carcinoma General. Since the days of Krompecker it has been known that a proportion of basal-cell carcinomas are not pure basal cell tumors, from 10 to 15 per cent contain squamous-cell elements and are,

TABLE 4 DELAY INTERVAL COMPARED WITH SIZE OF LESION
(STUDY OF 186 LESIONS)

Type of Carcinoma	Delay Interval (Average in years)	Size of Lesion		
		Under 1 cm. (Per Cent)	2 to 3 cm. (Per Cent)	Over 3 cm. (Per Cent)
Basal cell	4.3	51	46.5	92.5
Basosquamous cell	3.6	18	50.0	32.0
Squamous cell	2.4	18	52.0	30.0

Table 4 Basal-cell carcinoma shows the longest average time-interval (4.3 years) between discovery of the tumor by the patient and request for treatment, while the basosquamous-cell carcinoma has a somewhat shorter delay interval (3.6 years) and the squamous-cell carcinoma has the shortest delay period of all (2.4 years). When the average delay interval is compared with the size of the lesion, a correlation between the delay interval and rapidity of growth is suggested. Not only is the delay interval longer for the basal-cell type but the lesions are smaller when the patients belatedly present themselves. The squamous-cell lesion is the most rapidly growing, the basal-cell the slowest, while the basosquamous-cell carcinoma occupies an intermediate position.

therefore, known as mixed-cell or basosquamous-cell carcinomas. In our series about 10.5 per cent are of the mixed cell type. The basosquamous-cell carcinoma is a metastasizing tumor but metastasis is observed much less frequently than in the squamous-cell carcinoma. An unselected group of twenty-four basosquamous-cell carcinomas was observed over a total period of sixty-two years (for the group) during which follow up no evidence of metastasis appeared.

Gross Appearance. There is no characteristic clinical appearance which enables differentiation of this lesion from

the pure basal-cell or squamous-cell carcinoma (see Figs. 65 and 66). When comparing the growth rate of the three types of epidermoid carcinoma, one finds that the basosquamous-cell carcinoma is a more rapidly growing tumor than the average basal-cell lesion, but definitely slower than the squamous cell (see Table 4).

scopist. Not infrequently these tumors contain basal cells which are distinctly larger than usual. As a rule, however the basal-cell element has the appearance of the usual variety of basal-cell carcinoma.

Cystic Basal Cell Carcinoma In the experience of this clinic, the multiple familial variety of cystic epithelioma has

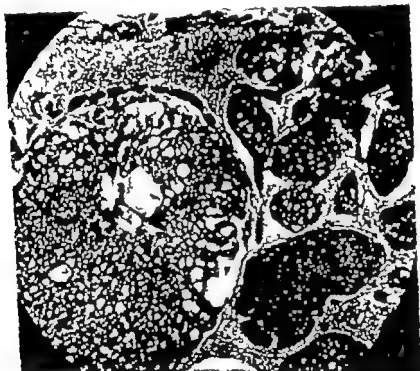


Fig. 67 Basal-Cell Carcinoma, Cystic Type. The cystic arrangement differs from Fig. 65, and the general pattern is that usually seen in the ordinary basal-cell carcinoma.

Hist pathology The tumor consists chiefly of basal cells with occasional nests or groups of squamous cells and scattered pearls, or whorls of varying frequency. In some cases the squamous-cell element is distinct and inescapable; in others, so slight as to be easily missed in a cursory examination. The possible occurrence of squamous-cell elements in a basal-cell tumor always should be remembered and excluded by the micro-

rarely been seen. The usual type of case presents a solitary lesion varying in size from that of a pea to 5 cm. or more in diameter. When small, the lesions are somewhat translucent. They are not slow growing and have a tendency to be invasive. The microscopic pattern is that of basal-cell carcinoma, but the cell strands or masses contain variable sized cysts with well defined linings. Some believe this lesion to be

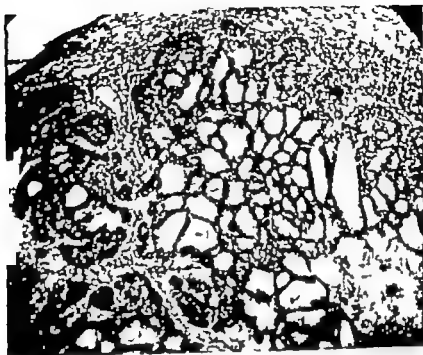


Fig. 68 Basal-Cell Carcinoma, Cystic Type. Note the morphous material in the cystic areas.



Fig. 69: *Left:* Syringocarcinoma. Of right cheek. *Basal-Cell Carcinoma. Of forehead.*
Right: Appearance after radium plaque therapy

a squamous-cell carcinoma (see Figs. 67 and 68). The contents of these cysts are said by some to be mucin. Occasionally seen are ordinary basal-cell carcinomas with cystlike spaces of necrosis. These should not be confused with cystic basal-cell carcinomas. They respond to irradiation as does the average basal cell lesion whereas the cystic basal-cell



Fig. 70 Syringocarcinoma. Adenocarcinoma of sweat gland near lateral ventricle in male age seventy-five. Lesion was widely excised after biopsy. Patient has been lost to follow-up.

carcinoma is ordinarily quite resistant to radiation.

Carcinoma of Appendiceal Structures of the Skin. Carcinomas arising from the appendiceal structures constitute probably less than 1 per cent of all skin cancers.

Sweat-Gland Carcinoma (Syringocarcinoma). This is an extremely uncommon tumor. Most carcinomas of sweat or sebaceous glands are nervous and relatively benign. It is likely that many cases of cylindroma are erroneously diagnosed as malignant carcinomas of the sweat gland; however true carcinomas of sebaceous or sweat glands do occur

Syringocarcinoma occasionally appears as a creamy white hard nodule under the skin and suggests, when seen early, a harmless sebaceous retention cyst. No diagnostic clinical features exist (see Figs. 69 and 70). There is a tendency to invade lymph nodes. The microscopic appearance is variable but tends toward the formation of alveolar structure and dilated ducts with associated papillary changes. Evidence of invasion should be seen (see Fig. 71).

Carcinoma in Sebaceous Gland. Sebaceous adenomas may occasionally be the seat of malignant degeneration. Typical appearing basal-cell carcinomas and adenocarcinomas have been seen. Some writers insist that diagnosis should be made only after microscopic serial section shows connection of the carcinoma with the mature glands.

Trichocarcinoma. Not uncommonly tumors which appear to originate in hair follicles are described. These tumors cannot always be positively identified. Metastasis appears to be uncommon (see Fig. 72).

Bowen's Disease, Paget's Disease and Cutaneous Metastatic Carcinoma Bowen's Disease. In 1912, Bowen separated this disease from other skin lesions and applied to it the designation precancerous dermatosis.

Various writers have aligned themselves on opposite sides in the controversy over the cancerous nature of Bowen's disease. In consideration of the views of most of the contemporary writers, we regard Bowen's disease as an intracutaneous carcinoma at the outset, rather than as a precancerous dermatosis. The lesion is not uncommon but may be confused clinically with the flat type of basal-cell carcinoma, the rare extramammary type of Paget's disease, lupus vulgaris, etc. (see Figs. 73 to 75).



Fig. 71: Syringocarcinoma. Note the variability of success in attempted differentiation into glandular pattern.

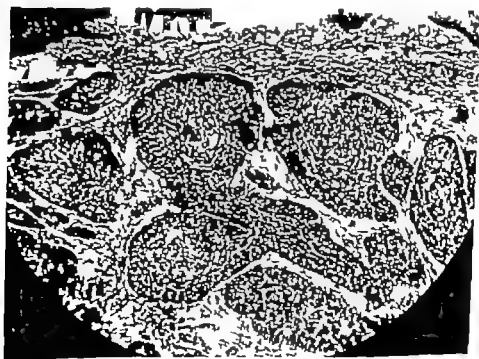


Fig. 72: Trichoepithelioma. The central portions of the cell nests suggest hair follicle structure. Note overlying epidermis.

CLINICAL APPEARANCE: Bowen's disease usually presents a flat, dull red, round or oval, well-defined blotch on the skin. Pruritus or pain may be present. The disease may start as a single patch or several closely grouped lesions which grow peripherally and eventually coalesce. The disease is unusually slow growing, but may on occasion produce metastasis.

HISTOPATHOLOGY Bowen's disease has been described as a basal-cell, mixed-cell,

mouth) characterized by its red color shiny papillomatous appearance and softness on palpation. It is resistant to all except destructive therapy. Sooner or later it undergoes malignant change with rapid draining lymph node metastasis. The histologic changes are not characteristic although the diagnostic changes seen in Bowen's disease (Darver) have been shown. It is considered by some to be an extramammary form of Paget's disease of the skin.

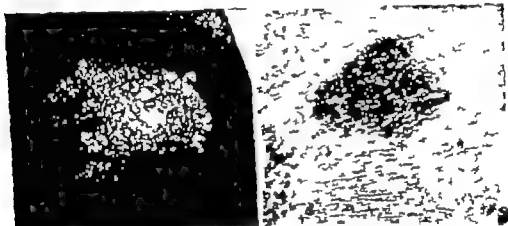


Fig. 73 Paget Disease (intraepidermal carcinoma) Of left shoulder

and squamous-cell carcinoma. Usually it is a squamous-cell lesion.

The microscopic picture is marked by hyperplasia of the epidermis, widening and elongation of the rete pegs, disorganization of the cells, intracellular edema, hyaline degeneration, and large hydropic giant cells (Bowen's cells or basket cells). There is eventual disruption of the basal-cell layer if invasive proliferation occurs. The corium shows perivascular small, round-cell, and plasma-cell infiltration.

Erythroplakia (Q erys) This is a precancerous condition. It is a chronic, circumscribed, but slowly extending papillomatous lesion of the demucosal and mucosal surfaces (glans penis, vulva,

Paget's Disease Paget, in 1874 described a specific disease of the mammary nipple. The true nature of the condition, now known as Paget's disease of the skin, has been in dispute ever since. Ewing regarded the lesion as precancerous, closely related to and invariably followed by carcinoma. Many others, notably among them Darver, Pautrier, Cheate, and Cutler, Massa and Rousset, and Broders, believe the disease to be carcinomatous from the start, intra ductal cancer or a squamous-cell cancer of the nipple itself. The controversy still exists and may be regarded as an unsettled question. Nevertheless, Paget's disease of the nipple unquestionably should

routinely be treated as a carcinoma of the breast. Paget's disease of the nipple properly falls in the domain of the breast surgeon or oncologist. Recurrent and certainly persistent eczema of the nipple

lesion in early Paget's disease of the nipple is atrophy of the nipple itself.

EXTRAMAMMARY PAGET'S DISEASE: This form of the disease has not been entirely accepted as a disease entity is uncommon, and appears to be improperly understood. Some cases clinically diagnosed as Paget's disease prove to be basal-cell carcinoma of the flat type, Bowen's disease, etc.

CLINICAL APPEARANCE: The disease is usually described as a solitary, red, eczematoid patch sharply delineated from the normal skin and varies considerably in size when first seen. The lesion may be dry and scaly or more usually intensely red, exudative, raw and granular. It is associated with tingling, itching or burning. Secondary infection is not uncommon. The site of predilection is stated to be the groin, scrotum, perineum, buttocks, and back.

HISTOPATHOLOGY: The characteristic feature is the Paget cell. Cheate states the diagnosis can be made by identification of Paget's cells from superficial scrapings. The Paget cell is present in greater numbers and is more typical in appearance at the spreading margins of the disease. This characteristic cell may be described as a large, hydropic, vacuo-



Fig. 74: Bowen Disease. A precancerous dermatosis.

and areola of the breast should be looked on with suspicion. Eczema of the nipple, however, is acute, has diffuse borders, and is commonly associated with scabies and the puerperal period. The essential



Fig. 75: Bowen Disease. A precancerous dermatosis.

lated, pale-staining, epithelial cell with out prickles. The hyperchromatic large nucleus is usually round but occasionally is lobulated. The Paget cell itself is considered by some to be malignant.

Cutaneous Metastatic Carcinoma. Metastatic carcinoma of the skin does



Fig. 76. *Cutaneous Metastatic Carcinoma.* Each circled area represents the site of cutaneous metastasis from primary carcinoma of the prostate. Each area treated with 750 of high voltage x-ray using 0.5 mm. Cu. filtration and 12 cm. diameter. Each pallid area denotes controlled all lesions until the patient's death. Few months later Fifty-nine such areas were treated, all of which are rapidly growing and extremely radiosensitive.

not present a characteristic clinical appearance. Ordinarily the multiplicity of lesions in addition to the history of a primary lesion of the breast, stomach, lung, or other organ makes identification easy (see Fig. 76). Lymphoblastomatous in-

filtration, etc. has to be excluded. A biopsy almost always establishes the metastatic nature of the skin condition and not infrequently aids in locating an unsuspected primary lesion. Microscopic differentiation from carcinoma of the dermal appendages should be made in solitary lesions. The lesions vary in size from a few millimeters to several centimeters and may be intracutaneous or subcutaneous.

Biopsy

The microscopic study of tissue specimens is of the utmost value in the diagnosis of cutaneous carcinoma and planning of treatment. Such a procedure is known as the "biopsy" and is indispensable in the evaluation of end results. It is generally agreed by men of experience that a properly performed biopsy of epidermoid carcinoma does not increase the danger of dissemination of the neoplastic cells. Recent controlled clinical experiments by Paterson and Nuttal and Mann and Unnig add further weight to this contention.

The biopsy may consist of complete and wide excision of the tumor followed by electrocoagulation or by primary closure. More frequently the biopsy is limited to partial excision of a small portion of the tumor.

The wedge-shaped biopsy is not recommended. A rectangular piece of tissue should be taken, three fourths of which is tumor tissue and one fourth of which is apparently normal tissue. In cases where ulceration is present, normal tissue is unnecessary if sufficient depth of tissue is obtained. In nonulcerated cases, if normal tissue is taken, the wound should be electrocoagulated immediately since the natural barrier to the carcinoma has been broken. Normal tissue is taken in the ratio of 1:4.

The aspiration biopsy which is now an accepted and valuable procedure, finds its usefulness in the evaluation of enlarged lymph nodes. It must be remembered however that a negative aspiration biopsy does not exclude the possibility of malignancy.

Partial biopsies are easily accomplished by sharp dissection or by the endotherm knife followed in each case by electrocoagulation. Aseptic precautions should always be observed in the preparation of the skin and in the surgical technique. Anesthesia is almost always by local infiltration. Care should be taken to avoid infiltrating the anesthetic solution into the tumor proper. Trauma by compression of the tumor before and after its removal should be avoided. The specimen which should be placed immediately in a good fixative (for example, 4 per cent formalin) must contain viable cells and should be large enough to satisfy the needs of the pathologists. Removal of bits of necrotic tissue or cutting of superficial specimens usually results in a negative or inconclusive pathological report. In most cases and particularly in early cases, the histological cellular relations are far more important than a cytologic study.

Radiation Physics

In the field of radiotherapy the rays at the command of the physician are a potent agent capable of doing much good if properly used but just as capable of doing injury if ignorantly employed. It is true that many more patients are harmed by undertreatment than by overtreatment but to give carcinocidal doses of irradiation demands of the physician that same degree of knowledge of radiation dosage and all possible effects as is required in prescribing ordinary potent medication. Only in this way can safe

adequate treatment be given and technique duplicated in the same or different clinics.

It is not within the range of this work to go into detail in the discussion of all the physical factors which are applicable in the irradiation therapy of carcinoma of the skin and its complications. Only the most relevant data pertaining to x rays and radium will be given herein as there are many excellent sources covering the subject of radiation physics.

X ray Physics

Dosimetry To describe accurately a given beam of irradiation or a roentgen-ray dose, both its intensity and quality at any given point in the beam must be known and must always be given. Intensity and quality will be discussed briefly in turn.

INTENSITY Intensity is described as exposure per unit of time or the amount of radiation traversing a given point during a specified time. It has nothing whatever to do with absorption, as we have no means of measuring actual absorption of irradiation in any given volume of tissue. The only property of roentgen rays or gamma rays which can be satisfactorily used to evaluate a physical dose which has a direct relation to tissue dose is the ability of a beam to ionize a gas, like ordinary air.

Unit of Dose the Roentgen The most recent definition (1937) of the roentgen modified so as to apply to x and gamma rays, reads as follows:

The roentgen shall be the quantity of x or gamma radiation such that the associated corpuscular emission per 0.001293 gram of air produces, in air ions carrying *i.e.* an of quantity of electricity of either sign.

All roentgen therapy apparatus when installed be it low intermediate or high

voltage, should have its output of irradiation competently calibrated in roentgens to cover all treatment techniques which will be used. Several reasons can be offered to show the necessity of determining intensity of output in roentgens. To begin with the variation in output between two identical installations or in the same unit at different times does not permit the mere statement of the tube potential, tube current, filter target-skin distance, field size and treatment time to represent the description of the dose. Furthermore the use of the erythema dose is unsatisfactory because writers often do not indicate whether reference is made to a threshold erythema or full erythema. In addition the evaluation of each is subject to wide individual variation. The error in dosage calibrations using this method may in some instances exceed 100 per cent. Finally if regression and total destruction of a tumor depend upon the delivery of a certain minimal dosage to every portion of the tumor and they do, then it is necessary to know the amount of irradiation delivered at various depths when treating fungating tumors, deep-seated tumors, metastatic nodes, etc. To obtain such knowledge of depth dose for any installation requires calibration of output in roentgens and the making of an absorption curve in order to obtain depth dosage data by comparison. It becomes obvious that output of irradiation must be calibrated in roentgens if scientific treatment is to be given with intelligence and confidence.

Units Fact Which Govern Quantity or Intensity of $\dot{V}R$ per The intensity in three situations is important, namely those of air, skin, and tissue. The factors which influence the intensity in these three locations will be discussed separately.

AS MEASURED IN AIR. This measurement excludes the back scatter of x rays from the tissues of the patient.

Voltage- As the voltage is increased, a greater number of roentgen rays of all wave lengths is produced, thereby increasing the intensity.

Milliamperage As more electrons are produced to bombard the target by an increase in filament current, there is a resultant increase in intensity.

Filter Inasmuch as the filter absorbs radiation passing through, the intensity is altered. The nature of characteristic irradiation produced in filters can be obtained from references in standard works on radiation physics.

Distance The intensity from a point source (the target may be considered as a point source) varies approximately as the inverse square of the distance (inverse square law). The greater the distance from target to patient, the less the intensity. As the T. S. D. (target skin distance) decreases, scattering from oil-immersed tubes modifies the law.

AS MEASURED ON THE SKIN OF THE PATIENT In addition to the previously mentioned factors, which always operate intensity as measured on the skin is modified by back scatter irradiation. A percentage of radiation entering a patient scatters back to the surface of the skin, thereby producing a greater intensity on the skin than in the air. The skin dose therefore, is the air dose plus the back scatter. Back scatter varies with the size of the field. In a small field there is less back scatter as the field size increases, the percentage of back scatter also increases. Back scatter also varies with the quality. One would anticipate greater back scatter from nonfiltered, low voltage x-rays (85 to 100 Kvp) than from filtered rays generated at a higher kilovoltage. Ac

tually this is not the case. Although it is true that a larger percentage of low voltage x ray energy penetrates only to the superficial layers, relatively more of these scattered rays are so weak that they are absorbed before they reach the surface of the skin to become back scat-

TABLE 5 BACK SCATTER FOR VARIOUS FIELDS AND QUALITIES OF RADIATION WITH THE PERCENTAGE OF PRIMARY BEAM AT 100 PER CENT*

Kv Peak	Filter Mm	H.V.L. Mm	Area of Field (Square Cm)			
			5	25	50	100
			Per Cent of Primary Beam			
120	H	1.0 Al	10	17	21	24
120	3.0 Al	4.1 Al	14	25	29	33
150	0.25 Cu	0.5 Cu	15	26	32	38
200	0.5 Cu	0.9 Cu	14	24	30	36
200	2.0 Cu	1.8 Cu	9	17	21	26

Courtesy of F. H. Quimby, Quimby Lectures, Ed and J. Brothers, 1911

ter irradiation. From this low point the percentage of back scatter increases as the voltage increases, reaching a maximum for 150 Kv p. irradiation filtered by 0.25 mm of copper. With filtered irradiation generated at 150 Kv p. or over the higher the half value layer (see "Quality") the less the back scatter (see Table 5). A certain depth of underlying tissue is required to produce maximum scattering and back scattering at a given Kv p.

TABLE 6 THRESHOLD ERYTHEMA DOSES FOR DIFFERENT QUALITIES OF IRRADIATION*

Kv	Filter Mm	H.V.L. Mm	Röntgens (At)	Röntgens (i Al)
50	0	0.3 Al	180	1.5
100	0	1.0 Al	225	1.5
140	4 Al	0.4 Cu	400	5.25
200	0.5 Cu	0.9 Cu	525	6.75
700	7.0 Cu	7.0 C	700	800

Courtesy of E. H. Quimby
† Small field on forearm.

TABLE 7 DEPTH DOSEAGE. HIGH VOLTAGE X RAY THERAPY OF SKIN CANCER (200 Kv $\frac{1}{2}$ Mm Cu + 1.0 Mm Al H.V.L. = 0.52 Mm Cu)*

Depth Cm	2" Cm. T.S.D				3 1/2 Cm. T.S.D			
	2 Cm. Circle		3 Cm. Circle		5 Cm. Circle		2 1/2 Cm. Circle	
	D.D	T.D	D.D	T.D	D.D	T.D	D.D	T.D
0	100	111	100	117	100	125	100	115
1	77	86	81	94	87	109	80	92
2	63	68	66	77	73	91	66	75
3	47	53	51	60	59	74	51	59
4	37	41	41	48	46	62	48	48
5	29	33	32	37	39	48	33	38

Courtesy of Pack and Wuester from Pack and Livingston Treatment of Cancer and Allied Disease, Paul B. Hoeber New York.

TABLE 8 TISSUE ROENTGENS FOR CORRESPONDING AIR ROENTGENS AT VARIOUS DEPTHS
IN THE TISSUE FOR DIFFERENT SIZE FIELDS.
(200 Kv $\frac{1}{2}$ Min. Cu Filter 40 Cm. STD 0.2-0.25 Min Cu H.V.L.)
100 Air Roentgens*

Depth Cm.	1.4	3.0	4.0	5.0	7.5	10	12.5	15.5										
	Cm.	Cm.	Cm.	Cm.	Cm.	Cm.	Cm.	Cm.										
	Do	Do	Do	Do	Do	Do	Do	Do										
	4 5				6 7.5	7.5	10	10	10	10	15	15	15	20	25	30	35	40
	Cm.				Cm.	Cm.	Cm.	Cm.	Cm.	Cm.	Cm.	Cm.	Cm.	Cm.	Cm.	Cm.	Cm.	Cm.
	4	7.5	12.6	20	40	72	100	120	120	225	300	300	300	400	400	400	400	400
	Cm	Cm	Cm	Cm	Cm	Cm	Cm	Cm	Cm	Cm	Cm	Cm	Cm	Cm	Cm	Cm	Cm	Cm
Surface	114	118	119	120	121	122	123	124	125	126	127	128	129	130	131	132	133	134
1	93	100	103	105	111	112	113	114	115	116	117	118	119	120	121	122	123	124
2	75	82	86	89	97	101	104	107	110	113	116	119	122	125	128	131	134	137
3	63	69	73	77	84	88	92	96	100	104	108	112	116	120	124	128	132	136
4	54	59	63	67	73	77	81	85	89	93	97	101	105	109	113	117	121	125
5	47	51	55	59	64	68	72	76	80	84	88	92	96	100	104	108	112	116
6	41	45	49	53	57	61	65	69	73	77	81	85	89	93	97	101	105	109
7	36	40	44	48	52	56	60	64	68	72	76	80	84	88	92	96	100	104
8	31	35	39	43	47	51	55	59	63	67	71	75	79	83	87	91	95	99
9	27	31	35	39	43	47	51	55	59	63	67	71	75	79	83	87	91	95
10	24	28	32	36	40	44	48	52	56	60	64	68	72	76	80	84	88	92
11	21	25	29	33	37	41	45	49	53	57	61	65	69	73	77	81	85	89
12	19	23	27	31	35	39	43	47	51	55	59	63	67	71	75	79	83	87
13	17	21	25	29	33	37	41	45	49	53	57	61	65	69	73	77	81	85
14	15	19	23	27	31	35	39	43	47	51	55	59	63	67	71	75	79	83
15	14	18	22	26	30	34	38	42	46	50	54	58	62	66	70	74	78	82
16	13	17	21	25	29	33	37	41	45	49	53	57	61	65	69	73	77	81
17	12	16	20	24	28	32	36	40	44	48	52	56	60	64	68	72	76	80
18	11	15	19	23	27	31	35	39	43	47	51	55	59	63	67	71	75	79
19	10	14	18	22	26	30	34	38	42	46	50	54	58	62	66	70	74	78
20	9	13	17	21	25	29	33	37	41	45	49	53	57	61	65	69	73	77

Courtesy of J. L. Weathermon.

In summary intensity as measured on the skin is governed by voltage, milliamperage, filtration, distance, size of portal, back scatter and depth of underlying tissue.

AS MEASURED IN TISSUE BENEATH THE SURFACE OF THE SKIN. A greater depth dose can be obtained by increasing voltage, increasing filtration, increasing skin target distance and increasing size of the field. The depth dose is also controlled by thickness of underlying tissue.

When a large portal of entry is employed, the percentage depth dose is greater than when a small field is used. With the voltage, filter and distance held constant, the greater the dimensions of the field the greater is the volume of tissue irradiated, with a resultant increased depth dose due to increased scattering of radiation in a larger volume of tissue. The percentage of depth dose also varies directly as the distance. As the target skin distance increases, with

TABLE 9 COMPARATIVE DEPTH DOSES OF DIFFERENT TYPES OF IRRADIATION
SKIN DOSE (SURFACE DOSE) EQUALS 100 PER CENT OR R*

Type of Irradiation	Filter	Distance	Size of Portal	Surface	1 Cm. Depth	2 Cm. Depth	3 Cm. Depth	4 Cm. Depth
200 kvp	0.5 mm. Cu	50 cm.	4 x 5 cm	100% or r	80 r	67 r	56 r	46 r
200 kvp	0.5 mm. Cu	25 cm.	4 x 5 cm	100% or r	85 r	69 r	59 r	49 r
140 kvp	0.25 mm. Cu	20 cm.	4 x 4 cm.	100% or r	87 r	70 r	58 r	48 r
150 kvp	1.0 mm. Al	23 cm.	4 x 5 cm.	100% or r	81 r	63 r	49 r	38 r
150 kvp	Inherent	23 cm.	4 x 5 cm.	100% or r	75 r	51 r	37 r	28 r
100 kvp	1.0 mm. Al	23 cm.	4 x 5 cm.	100% or r	81 r	64 r	47 r	35 r
100 kvp	Inherent	23 cm.	4 x 5 cm.	100% or r	70 r	48 r	33 r	25 r
60 kv	0.2 mm. Ni	5 cm.	18 sq cm	100% or r	68	44 r	28 r	18 r
(Chaoel)								
50 kv	0.2 mm. Al	4 cm.	5.5 sq cm.	100% or r	45 r	21 r	15.5 r	8.4 r
(Phillips)								
Rad um	0.5 mm. Pt	3 cm.	20 sq cm.	100% or r	67 r	46 r	30 r	23 r
Radium	0.5 mm. Pt	2 cm.	20 sq cm.	100% or	61 r	41 r	30 r	21.3 r
Radium	0.5 mm. Pt	1 cm.	20 sq cm	100% or r	52 r	32 r	21 r	14.8 r
Radium	0.5 mm. Pt	0.5 cm	20 sq cm	100% or r	45 r	25	16	11.3 r

Courtesy of J. L. Weatherwax (Modified by S. C. C.)

other factors held constant, the percentage depth dose increases. This is explained on the basis of a narrower beam and a consequent lesser geometrical spread of the rays. The percentage depth dose also varies with the quality of irradiation. As the half value layer increases, that is to say, as the x ray beam becomes harder, the percentage depth dose also increases (see "Quality").

Tissue Dose. This may be defined as the exposure or dose at a specified point either on the surface or any given depth in tissue. Tissue doses are usually expressed either in terms of roentgens or threshold erythema doses (T. E. D.). The number of roentgens which will produce an erythema varies with the quality of the radiation, size of portal, area of body impression of observer, etc. (see Table 6). Values for T. E. D. also vary considerably in different clinics.

Depth Dose or Depth Intensity Charts. These are used to show graphically the percentage of irradiation (tissue dose)

reaching various depths as compared to 100 per cent on the skin or in air. Such data have been obtained for various sized fields and for different qualities of irradiation. Two types of depth dose charts exist: (a) those which show dose only along the axis of the beam (see Tables 7 and 8); (b) iso-dose curves which in addition give the dose at points off the axis of the beam. Fig. 77 shows iso-dose curves for various sized portals for 140 Kvp radiation with a technique similar to that used in the treatment of epidermoid carcinoma at the American Oncologic Hospital. Weatherwax has compared at 1, 2, 3 and 4 cm depth the tissue dose of various types of irradiation (see Table 9). These data are of especial value to those treating carcinoma of the skin.

QUALITY. The roentgen deals with quantity. Quality deals with the penetrating power of the rays. The harder or more penetrating a beam of x rays, the shorter is its average wave length, the

Physical Investigations with 140 kVp Radiation

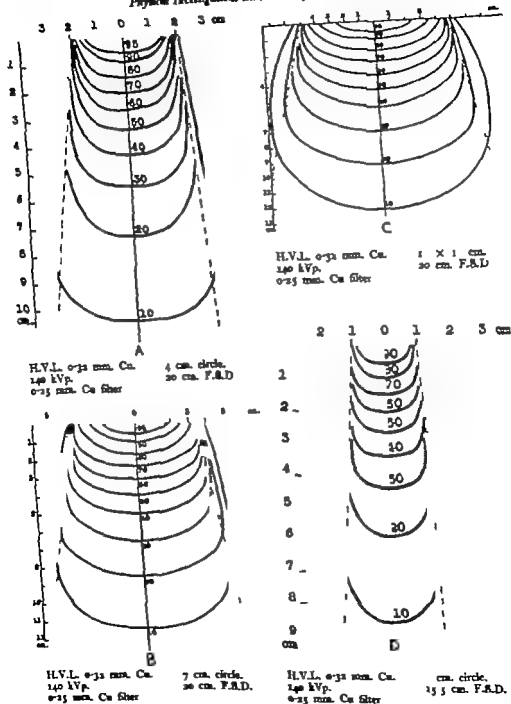


Fig. 77 (Permission of L. F. Lamerton. Courtesy of Brit. J. Radiol., 1941.)

softer a beam the longer is its average wave length. A beam of roentgen rays coming directly from the tube consists of a mixture of rays of different wave lengths varying from the shortest that can be produced from a given kilovoltage to the longest which can escape through the tube therefore these rays have a wide range of penetrating power. The only way to define such a beam completely is to determine its spectrum (by means of a spectrometer) and by determination of the intensity for each wave length. Inasmuch as such a method is not practicable except in research laboratories simpler methods usually are used in order to obtain information concerning the quality of roentgen irradiation which is sufficiently accurate for clinical purposes.

Various methods have been used to describe quality. The half value layer (H V L) is the most widely used method and is approved by international committees. To obtain the half value layer the construction of an absorption curve is necessary.

The H V L may be defined as the thickness of some standard material for instance aluminum or copper necessary to reduce by half the intensity of a given beam of roentgen irradiation. The harder the beam or the shorter the wave length the more penetrating are the rays and naturally it follows the greater is the thickness of material required to reduce the intensity by one-half. Now it is obvious that the half value layer increases as the rays are made harder and more penetrating. Half value layers of radiation between 20 and 130 K.v.p. are expressed in aluminum between 130 and 400 K.v.p. in copper. The factors governing the quality of x rays in air are

Target Material. The characteristic x ray wave lengths vary with the atomic

weight. General or background x ray wave lengths are the same for any target material and vary with the voltage.

Voltage. As the voltage is increased, the minimum wave length becomes shorter. The rays may be said to become harder—more penetrating.

Filter. A filter removes a portion of all wave lengths but relatively less of the shorter wave lengths, consequently the addition of filtration is said to improve the quality of the rays with a resultant harder beam. As the thickness of the filter increases, the percentage of absorption increases until eventual total or 100 per cent absorption occurs. In higher voltage therapy characteristic radiation which develops in primary filters is absorbed by the use of secondary filters of lower atomic weight. The soft characteristic irradiation from secondary filters is insignificant.

Methods of Application of Roentgen Rays

Contact Therapy (by Methods of Chaoul and Phillips). This method is not widely used. It is likely that these units have a little or no replacement value over apparatus such as the ordinary low or moderate voltage x ray units, at least so far as treatment of epidermoid carcinoma of the skin is concerned.

XV 45 to 60

FILTER. 0.2 mm of Al to 0.2 mm copper plus 1 mm Al or nickel.

DISTANCE. 2 to 5 cm

DOSE. 6000 to 10 000 r or more delivered in daily fractions of 500 to 1000 r or more.

Low Voltage (Superficial) Technique. Best used for keratosis or superficial flat epidermoid carcinoma. May be satisfactorily used for the treatment of lesions previously modified (flattened) by surgery.

Fractional. *KVP* 55 to 100

FILTER Inherent to 2 mm Al.

Distance 10 to 20 cm

ROENTGENS 500 r daily or every other day to totals of 4000 r to 7000 r or more (see Table 9)

Massive *KVP* 85 to 100

FILTER Inherent to 6 mm Al or $\frac{1}{4}$ mm Cu plus 1 mm Al

Distance. 10 to 20 cm

ROENTGENS 1000 to 6000 Massive doses of the lower order of magnitude are repeated either daily every other day or once a week to totals of 3000 to 6000 r or more. Doses of 6000 r in one sitting should never be delivered to a field over 1 cm in diameter. Such massive doses are not ordinarily recommended.

Intermediate Voltage Technic Satisfactory for most epidermoid carcinoma where irradiation is indicated.

Fractional. *KVP* 130 to 140

FILTER 4 to 6 mm Al or $\frac{1}{2}$ mm Cu.

Distance 10 to 50 cm

ROENTGENS 500 r per exposure daily or very other day or every third day. Total quantity of radiation varies from 3500 to 6000 r or more depending on daily dose, total time of treatment, portal size, depth of tumor etc. (see Table 9)

Massive *KVP* 130 to 140

FILTER 4 to 6 mm Al or $\frac{1}{2}$ mm Cu plus 1 mm Al

Distance 10 to 20 cm

ROENTGENS 1500 to 3000 given in single exposure. Tissue reaction depends on size of field which includes a relatively small border of normal skin. The lower doses are sometimes used in conjunction with electrosurgical excision. This method of treatment is not recommended.

High Voltage Technic (Deep Therapy) Fractional. Usually employed for large infiltrating epidermoid carcinoma, skin cancer involving cartilage, recurrent lesions or metastatic lesions.

KVP. 180 to 220

FILTER 0.5 mm. to 2.0 mm Cu plus 1 mm. Al or Thoraeous filter (0.42 mm Sn 0.25 mm Cu, 1 mm. Al) The filtration usually is 0.5 mm. Cu to 1 mm. Cu

Distance. 30 to 50 cm.

ROENTGENS Daily exposures of 200 to 500 r. Treatment totals are carried from 3600 to as high as 8000 or more roentgens if relatively small portals are used.

Measure Has been used in conjunction with electrosurgical excision. Makes use of high voltage and low or moderate voltage x rays in one massive dose by delivering 750 to 1000 r of high voltage, followed immediately by 450 r of low or moderate voltage. Its good results were undoubtedly due to wide surgical excision. This technic is mentioned only to be condemned.

Radioactivity

A radioactive substance may be sufficiently well described as one which possesses the property of spontaneous emission of radiations capable of passing through metal and other light-opaque matter. There are a number of radioactive substances known. Only the radium series of the uranium family of radioactive elements is of clinical importance; therefore the discussion will be limited to radium.

The radiations from radium are of two distinct types: that is, corpuscular and electromagnetic. The corpuscular rays are the so-called alpha and beta rays. The alpha rays may be described as positively charged particles having a mass four times that of the hydrogen atom and are identical with the helium nucleus. The corpuscular beta rays are fast moving negative electrons, similar to the cathode particles or rays of an x ray tube. The important penetrating electromagnetic gamma rays of radium have no mass.

TABLE 10 RADIUM AND ITS DISINTEGRATION PRODUCTS*

<i>Element</i>	<i>Atomic Weight</i>	<i>Atomic Number</i>	<i>Type of Radiation</i>	<i>Half Value Period</i>	<i>Transformation Constant</i>
Radium	226	88	alpha	1580 years	1.57×10
Radon	222	86	alpha	3.824 days	2.1×10
Radium A	218	84	alpha	3.05 minutes	3.79×10
Radium B	214	82	beta gamma	20.8 minutes	4.31×10
Radium C	214	83	alpha beta gamma	19.7 minutes	5.86×10
Radium D	210	82	beta gamma	22 years	1.57×10
Radium E	210	83	beta gamma	4.8 days	1.00×10
Radium F (polonium)	210	84	alpha	140 days	3.89×10
Radium G (uranium lead)	206	82	stable		

Courtesy of J. Chadwick Pittman and Sons.

and no charge they have the same physical properties as x rays, but are more penetrating.

The Transformation Theory According to the theory of Rutherford and Soddy which was advanced to explain the phenomenon of radioactivity the atoms of a radioactive substance undergo an orderly process of spontaneous disintegration which gives rise to the formation of new atoms, distinct from the parent in physical and chemical properties. This spontaneous decay is uncontrollable and completely unaffected by physical or chemical action. About one atom in 10^{21} breaks up each second; half the atoms of an amount of radium in about 1600 years.

The decay is the result of the emission of alpha and beta particles from an atom (see Table 10). With the emission of alpha and beta particles, a new atom itself unstable is formed.

From the disintegration of uranium

the element radium is formed which, by giving up an alpha particle forms radon, an inert radioactive gas which can be collected by physical means and is of great therapeutic value. Radon breaks down and successively radium A, B and C are formed. Radium C, for all practical purposes, can be said to represent the source of gamma and beta radiation used clinically. The disintegration continues from radium C through a number of definite stages leading eventually to the formation in forty years of stable radium lead with an atomic weight of 206. All elements with an atomic weight over 206 are radioactive (see Table 10).

The Half Life The length of time for any member of the radioactive series to disintegrate to one-half of its original amount is called the "half life period" or merely the "half period." Half of any remaining amount disintegrates in the same length of time required for the dis-

integration of the first one half Table 10 shows the half-life of radium to be 1580 years and, for radon, 3.82 days. The half life period is not synonymous with and should not be confused with the average life period.

The Average Life The average life is that hypothetical period which would exist if the radiation were emitted at a constant rate until it suddenly ceased to exist. The average life is the product of the half life multiplied by the factor 1.43. This factor is of value in calculating the total expected dose from the disintegrating radon. The average life for radon is 5.53 days or 133.3 hours.

Penetration through Matter α rays, alpha, beta, and gamma rays are all capable of producing comparable biological effect in tissue by means of a physical characteristic which is common to each, namely the direct or indirect ionization of matter. The feeble alpha rays, which can be completely absorbed in a few centimeters of air or by a sheet of writing paper have doubtful clinical import. The beta rays vary in penetrating power but are practically all absorbed by 0.5 mm. platinum or 1 mm. of lead. Radon bulbs or radium element needles, with a wall thickness considerably less than 0.5 mm. platinum are used when beta radiation is desired for clinical use.

The powerfully penetrating gamma ray, the most frequently used radiation of radium, can penetrate many inches of lead. The ionizing radiation from radium may be employed in two different forms, that is, in the solid form of a radium salt, or in the gaseous form of radon. Radium, in its form of a solid is placed in cells

and fitted in permanent needles. Because of the long half-life (1580 years) the radiations emitted from such a container are considered as constant for any lifetime. Radon, on the other hand after reaching its equilibrium amount, decays to one half its original radioactive strength in 3.82 days.

Equilibrium amount may be defined as the maximum amount of any radioactive element which can be formed from a given amount of its parent. Because of the rapid decay of radon and its separation from its parent a different method which will be described presently is employed in dosage computations.

Radium Dosimetry

Primarily the intensity of radiation from a quantity of radium depends on its amount or weight. A radium standard of known weight is needed to calibrate unknown amounts of radium. This determination is done by means of an ionization chamber. In 1912, Madame Curie's standard of very pure radium chloride, weighing 21.09 mg. was adopted as the international standard. The U. S. Bureau of Standards, in 1913, secured a secondary standard weighing 11.44 mg.

Methods of Recording Dosage or Exposure *Milligram Hours* This expresses the dose in terms of the amount of radioactive material used and the length of the period of treatment. For example one milligram of radium employed in treatment for one hour represents a dose of one milligram hour. This was the universal method of radium dosimetry. With a given size and shape of applicator, thickness of filter and quantity of radium, a definite known quantity of energy reaches the source; however the expression in milligram hours remains the same regardless of distance of the source from the treated area. The inadequacy

The alpha ray in radon treatment is considered by some observers to be the chief therapeutic agent. It is used in the treatment of chronic radiation ulcerations. Others believe the beta ray to be the dominant effective agent.

of this method of dosage specification is at once seen as it states the dose merely at the source nothing is known of the dose reaching the tissue. This method of dosage specification nevertheless is still widely used.

Millicurie Hours A curie is defined as that amount of radon which is in equilibrium with 1 gm of radium in other words that amount which has the same gamma ray activity as 1 gm of radium. A millicurie represents one-thousandth of a curie. The curie is the standard method of dosage specification for radon. This method also fails to provide information concerning irradiation reaching the tissues.

Millicurie Destroyed Inasmuch as radon is constantly and rapidly breaking down treatments of more than a few hours, where radon is employed must take into consideration the constantly decreasing amount. One millicurie of radon during its entire life will deliver 133.3 mc hours or the same amount delivered by 1 mg of radium in 133.3 hours, therefore the total amount of radiation emitted by 1 mc of radon during its entire life is designated as 1 millicurie destroyed (written mc d). This method of dosage calibration has been widely used in France to describe radium as well as radon dosage. This method likewise provides no information concerning tissue dose.

Threshold Erythema Dose The T E D is based on the number of milligram hours millicurie destroyed or roentgens which will produce a faint blush within a week or ten days in about 80 per cent of cases and will have no visible effects on 20 per cent. Using radium filtered with 0.5 mm platinum the dose is approximately 1000 r. The dose in milligram hours varies with the filter distance, and size of applicator.

Equivalent Roentgens Various efforts to describe radium dosage in such a manner as to give information concerning the radiation actually reaching the skin have been efforts to overcome the deficiencies of dose designation as used at present.

Mayneord and others have attempted to measure milligram hours in terms of gamma roentgens. One mg of radium from a point source at 1 cm distance and filtered with 0.5 mm of platinum will give a value of 8.3 r in one hour. Patterson and Parker have correlated milligram hours for different types and sizes of radium applicators in terms of roentgens at various distances. These methods are not employed as widely as they should be.

Radium Dose in Tissue As has been stated the milligram hour and millicurie destroyed as expressions of dose, are concerned only with the radiation at the source. The therapist should be more concerned with the amount of radiation reaching the tissue at the surface and at various depths. The dose reaching the tissue from an amount of radium depends on (1) the size and shape of the applicator (2) the filtration and (3) the distance. Various tables are available which give information concerning these factors.

Size of Applicator As the size of an applicator containing a given amount of radium is increased the number of mg hrs. necessary to deliver a threshold erythema dose also increases, however as the size of the applicator is increased the percentage depth dose is greater.

Filter The alpha particles are removed by ordinary paper or 0.002 mm of aluminum. Two mm of brass or its equivalent of 0.5 mm of platinum are required to remove all the beta radiation (see Fig 78). After removal of all the

caustic beta radiation, little is gained by further filtration. A secondary filter should be used between applicator and skin to absorb characteristic irradiation of the filter. A few millimeters of rubber suffice.

Primary filters of various metals, such as monel metal silver gold, and platinum are used. Secondary filters of brass and

arable damage to the intervening tissue. In such instances, the implantation of either gold or platinum radon seeds or radium needles directly into the diseased mass is advised hence, because of the rapid loss of intensity as the distance from the source increases, it is possible to deliver relatively high doses (several additional erythema doses) to the dis-

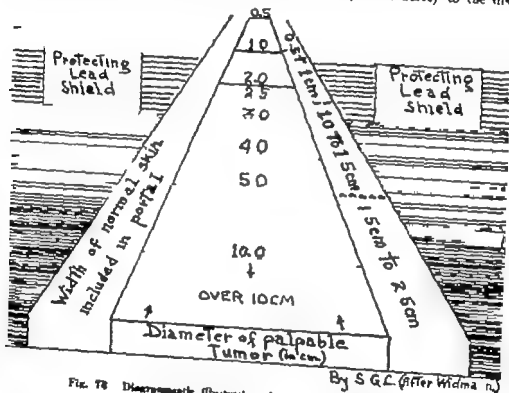


FIG. 78 Diagrammatic illustration showing width of normal skin which should be included in the treatment portal.

lead are also used. The filtration equivalents of the various filters are computed by comparison of their specific gravities.

Distance: The intensity of radiation fall off rapidly as the distance from the source is increased due to divergence of the rays. This is shown in Table 9.

Interstitial Radiation: It is often impossible to deliver by external irradiation alone an adequate dose to a tumor such as a metastatic node without irrepar-

able damage to the intervening tissue without undue damage to the surrounding normal tissue.

For purposes of dosage calculation when employing radon seeds, lesions are considered as spheres or groups of spheres. When radium needles are used, the problem is rendered more complex by variation of filter needle length etc. The needles are sometimes distributed in planes. The original tables, charts, etc., should be consulted.

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Threshold Erythema Dose The T E D is based on the number of milligram hours, millicurie destroyed or roentgens which will produce a faint blush within a week or ten days in about 80 per cent of cases and will have no visible effects on 20 per cent. Using radium filtered with 0.5 mm platinum the dose is approximately 1000 r. The dose in milligram hours varies with the filter distance, and size of applicator.

Equivalent Roentgens Various efforts to describe radium dosage in such a manner as to give information concerning the radiation actually reaching the skin have been efforts to overcome the deficiencies of dose designation as used at present.

Mayneord and others have attempted to measure milligram hours in terms of gamma roentgens. One mg of radium from a point source at 1 cm distance and filtered with 0.5 mm of platinum will give a value of 8.3 r in one hour. Latterson and Parker have correlated milligram hours for different types and sizes of radium applicators in terms of roentgens at various distances. These methods are not employed as widely as they should be.

Radium Dose in Tissue As has been stated the milligram hour and millicurie destroyed as expressions of dose, are concerned only with the radiation at the source. The therapist should be more concerned with the amount of radiation reaching the tissue at the surface and at various depths. The dose reaching the tissue from an amount of radium depends on (1) the size and shape of the applicator (2) the filtration and (3) the distance. Various tables are available which give information concerning these factors.

Size of Applicator As the size of an applicator containing a given amount of radium is increased the number of mg hrs necessary to deliver a threshold erythema dose also increases; however as the size of the applicator is increased the percentage depth dose is greater.

Filter The alpha particles are removed by ordinary paper or 0.002 mm of aluminum. Two mm of brass or its equivalent of 0.5 mm of platinum are required to remove all the beta radiation (see Fig 78). After removal of all the

of the various cells has been graded by Desjardins as follows.

Lymphoid cells most sensitive (least radio-resistant)

Epithelial cells, basal cells of skin.

Endothelial cells.

Connective-tissue cells.

Muscle cells.

Bone cells.

Nerve cells, least sensitive (most radio-resistant)

In general, there is a fair degree of correlation between the degree of sensitivity of a neoplasm and the tissue from which it develops. In other words, if the adult tissue is radiosensitive a tumor originating in that tissue ordinarily but not always is expected to be radiosensitive. In consideration of the embryonal nature of the cancer cell one would quite naturally expect a greater degree of sensitivity in the neoplastic cell springing from those of any origin. This is generally true and upon this premise the success of general radiotherapeutics is founded.

It should be obvious that a tumor which is decidedly more radiosensitive than the normal adult tissue forming the tumor-bed should and can be destroyed with nonlethal damage to the surrounding tissue. Tumors which are relatively radio-resistant when compared to tissues of the tumor-bed can be successfully treated only if accessible. Into this category fall many indeed most skin cancers. Carcinocidal doses are possible only because of the favorable anatomic situation of the majority of these lesions.

The radiosensitivity of a cell is not a static constant phenomenon, but is alleged to vary according to whether a cell is actively dividing or is in the resting stage. In this connection, however, experience shows that not infrequently tumors which show active mitoses and are thought to be radiosensitive not infrequently fail to behave in the ex-

pected radiosensitive manner. Likewise many cornifying, apparently slow growing squamous-cell carcinomas reveal unexpected radiosensitivity during and following treatment. It can be stated that each variety of cell and tumor has a rather wide range of radiosensitivity.

RADIOSENSITIVITY OF SKIN CANCER

Resistant Group

Malignant melanoma	Extremely radio-resistant.
Squamous-cell carcinoma	The cells are undergoing rapid cell division but are relatively mature.
Trochocarcinoma	The cells are mature, differentiated and usually are not undergoing rapid cell division.
Syringocarcinoma	
Cystic basal-cell carcinoma	
Metastatic cutaneous cancer	Usually derived from primary carcinomas which are radio-resistant.

Sensitive Group

Lymphoblastoma	Extremely radiosensitive has first treated
Basal-cell carcinoma	Immature and undifferentiated cells which are relatively slowly but constantly multiplying.
Metastatic carcinoma of skin	Those derived from primary tumors which are radiosensitive, as some breast carcinomas, etc.
Squamous cell carcinoma	A small percentage is relatively sensitive.

The Carcinocidal Dose

A carcinocidal dose may be theoretically defined as that amount of irradiation delivered which will destroy every neoplastic cell in a local lesion, that is a tumor-sterilizing dose. This theoretical dose, if delivered, should permit of no recurrence.

Just what quantity of x-irradiation constitutes a carcinocidal dose for cutaneous carcinomas is a point of consid-

Armamentarium Radium needles or tubes of different intensity active length and filtration radon seeds, radon bulb, radium element pack or radium bomb.

Protection Anyone who employs x or gamma irradiation should be completely familiar with its potential dangers to the patient, technician and therapist. This knowledge is easily obtainable from handbooks published by the U. S. Department of Commerce.

Maintenance of Records The therapist should include in his treatment records data that will permit him at some future date to evaluate for himself or to convey to anyone the exact technique employed in the treatment of a lesion.

X Rays For x rays the data should include

OPERATIONAL FACTORS

- 1 Voltage
- 2 Milliamperage
- 3 Filter
- 4 Target skin distance
- 5 Exact (measured) size of portals
- 6 Roentgen per minute (as measured in air)
- 7 Half value layer
- 8 Effective wave length (optional)

DOSAGE TECHNIC

- 1 The number of roentgen given at one treatment to one or more fields. (Anatomical charts for location of field should be used.)
- 2 Total roentgens (as measured in air and on skin)
- 3 The dates of each treatment
- 4 Total daily treatment time
- 5 The duration of the completed series.

RADIATION DISTRIBUTION IN PATIENT (Applies Only to High Voltage Therapy)

- 1 Total roentgen to each portal (measured in air)
- 2 Total roentgens to each portal (skin dose)
- 3 Depth or tissue-dose from each portal
- 4 Total tumor-dose by summation of the tissue-dose of two or more fields employed in cross fire

Radium. For radium the data should include

- 1 Quantity of radium or radon used in applicator
- 2 Filter
- 3 Distance
- 4 Number of tubes or needles used, size of applicator (including active length of needles)
- 5 Diagram of applicator showing distribution of needles.
- 6 In interstitial work the dimensions and volume of tissue to be treated should be calculated. Sketches should be used to show position of implants.

Biological Effects of Radiation with Particular Reference to Skin

The action of ionizing irradiation on living tissue is always destructive at least in degree; the degree varies in an irradiated cell dependent upon the dose. In a volume of tissue following a certain moderate dosage of irradiation there may be found damaged cells which will recover, damaged cells which will go on to death after a period of time, and damaged cells which will probably die almost immediately. To account for the various degrees of damage, many complex and incompletely understood phenomena have been described.

For practical purposes it may be stated that the changes which result from ionizing irradiation in any cell are probably the result of a direct action on the cell, an indirect action on the cell by the adverse modification of its environment in some manner, or a combination of both actions. The radiation effect depends on the intensity of the applied stimulus, radiosensitivity of the cell, and the environment of the cell.

Radiosensitivity The different normal tissues in the human body vary in degree of radiosensitivity that is to say the various tissues require different dosages of radiation to effect destructive changes in the cell. The radiosensitivity

ment of some radiosensitive lesions is essential to assure adequate treatment for all epidermoid carcinoma, many of which are relatively resistant. Such a method of treatment, if followed by all therapists, will effect marked improvement in the end results of epidermoid carcinoma about the face.

Quality of Irradiation and Carcinocidal Dose Upon this question authoritative opinion varies widely but it appears that the difference which exists when comparing carcinocidal doses delivered from 100 K.v.p., 140 K.v.p. or 200 K.v.p. units may be explained by known physical data.

It is true that when low voltage irradiation is used, greater absorption of irradiation energy takes place in the more superficial tissues. In view of this fact, it would be expected that in relatively flat lesions, the quantity of radiation to produce a tumor-sterilizing dose should be somewhat less for superficial therapy (90 to 100 K.v.p.) than for higher voltage therapy (140 to 200 K.v.p.). Actually this does not appear to be the case. The better spatial distribution and more uniform beam of filtered higher voltage x-ray brings the carcinocidal dose to a level somewhat lower than that of low voltage x-rays. When thick lesions are treated, obviously the dose using higher voltage x-rays is much lower than the dose required from low voltage rays.

Carcinocidal Dose (Radium) Little has been said about the carcinocidal dose of gamma rays. Because of the marked variation in technique from clinic to clinic little may be said. Most observers use the threshold erythema as a measure of dose specification. The carcinocidal dose for squamous-cell cancer is said to range from 7 to 12 or more T. E. D. Pack states that the dose to

destroy the most radioresistant epidermoid carcinoma varies from 0.5 to 3 mc destroyed, or 75 to 400 mc or mg hrs per sq cm of surface irradiated. The dose, of course, varies between limits dependent on surface area, thickness of the tumor, distance filtration, etc. In our clinic, we seldom treat lesions which are over 3 cm in diameter with surface radium. The dose varies from 75 to 150 mg. hrs per sq cm., using 0.5 mm. monel metal filtered needles at direct contact for flat lesions, to 400 to 600 mg. hrs. per sq cm at 1 cm distance, using the equivalent of 0.5 mm. of platinum-iridium filtration for elevated lesions.

Little need be said here concerning the carcinocidal dose as delivered from a radium bomb. Few such units are in operation and are seldom used in the treatment of skin cancer except in occasional far-advanced cases or in the treatment of metastases. Dosage is a variable matter individualized in different clinics because of such factors as distance, radium-needle arrangement, etc.

Tissue Tolerance Dose

During the irradiation of tissue, a point can be reached where any added amount of irradiation will produce an irreversible reaction which continues to the point of tissue destruction and which may be permanent. There is, therefore, a tissue-tolerance dose. Precisely what this dose is in any given case cannot be predetermined. The tolerance dose depends on many factors: (1) previous treatment, (2) fractionation, (3) portal size, (4) quantity of irradiation per unit time, (5) quality of rays, (6) the underlying tissue, (7) the presence of infection, (8) individual hypersensitivity, etc.

Doses as high as 10,000 r (no filter) have been delivered to portals 1 cm. in diameter without ill effects. In general,

erable contention. At the outset it should be mentioned that no fixed or constant dose actually can exist as the variation in radiosensitivity in the different types is quite considerable. For example, Widmann shows that using massive-dose technic given in one sitting 23 per cent of epidermoid carcinomas were controlled with 1000 r (2 S.F.D.) 47 per cent with 1500 r (3 S.F.D.) 78 per cent with 2000 r (4 S.F.D.) and 97 per cent with 2500 r (5 S.F.D.). An S.F.D. was established at 500 r using 195 K_vp with inherent filter. The variability of radiosensitivity of skin cancers is excellently drawn but the important fact brought out by Widmann's work is the necessity of empiricism in the successful radiation treatment for cancer of the skin that is a dose and technic should be adopted which experience has shown is able to destroy every neoplastic cell in the more radioresistant carcinomas. Such a dose should then be regarded as a working carcinocidal dose.

Never to be employed is a "hit and miss" treat-and-observe" technic which goes on until the lesion disappears or fails to respond further to the particular type of treatment. Such a method is followed by a high percentage of recurrence and generally unsatisfactory results as shown in the early experience of this and undoubtedly every clinic. Such a technic is condemned.

A so-called carcinocidal dose which varies with individual technic has been worked out for many different clinics. Depending on the size of the portal, quality of rays, fractionation and response of the lesion the dose varies from 3500 r to 7000 r or more usually fractionated over a period of one to two weeks (Chaoul and Philips technic excluded). Paterson predicts that the carcinocidal dose for a true squamous-cell

carcinoma will ultimately be from 5000 r delivered in one week to 6000 or 7000 r delivered in one or two months.

In our clinic, a dose of 3500 r* is regarded as *minimal* for any and every lesion many require more. The higher doses are reserved for some unusually resistant basal cell carcinomas, many squamous-cell lesions, and recurrent or primarily uncontrolled lesions. From 3500 r to 5000 r usually have been delivered in two to three weeks, using moderate voltage therapy and 5000 r to 6000 r usually have been delivered in two to four weeks, using high voltage irradiation. Rarely have we exceeded 6000 r to a 5 cm. circular portal, using moderate voltage (140 K_vp) x-rays. Widmann delivers 3200 r usually in one week with moderate voltage for flat lesions and 4800 r in one week for fungating lesions.

Dosage in the range of magnitude described by Widmann, or that employed in our clinic is not only safe but will destroy 95 to 97 per cent of all primarily treated epidermoid carcinomata of the face. If such dosage is insufficient to destroy the tumor surgical interference is usually indicated.

The ability of the therapist should not be judged by his disposition to carry doses to abnormally high levels, but rather by his enlightened willingness to carry out whatever treatment is best for his patient be it x-ray, radium, or surgery alone or in any combination.

It must be repeated that in the treatment of cancer of the skin a dose which will assure adequate treatment of the more radioresistant skin carcinomas should be selected. All skin cancers should receive that dose in spite of the fact that in a fair percentage of cases it might be unnecessary. Safe overtreat-

*As measured in air unless otherwise indicated.

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Doses as high as 10,000 r (no filter) have been delivered to portals 1 cm in diameter without ill effects. In general,

3500 to 4000 r (100 K.v.p.) in a single massive dose should not be given to cancers over 5 cm in diameter. With moderate voltage (130 to 140 K.v.p.) doses in the range of 2000 to 2500 r may be considered as safe given in a single massive dose. However there appears to be little reason for such massive doses. Fractionation will permit of considerably higher doses (see Treatment, below).

Large doses are better tolerated when delivered to a small volume of tissue. Inasmuch as the ability of tissue to regenerate decreases as the volume of tissue irradiated is increased the tissue tolerance decreases as the irradiated volume is extended therefore by restriction of radiation to carefully planned small but ample sized fields, higher doses can be delivered.

In larger fields over 5 cm in diameter if the tumor is fungating (2 to 3 cm thick) the total dose should not be reduced but should always be fractionated to assure safety. Where large flat lesions of 10 cm or more in diameter are to be treated total doses of 3000 to 4000 r using moderate voltage probably should not be exceeded. Small residual areas, however can be carried to higher doses (10 000 r or more) in exceptional cases.

Because of the decreased regenerative power of the tissue irrespective of whether superficial, moderate, or high voltage irradiation is employed repeated intensive courses of irradiation to the same field are dangerous.

Treatment of Epidermoid Carcinoma

Because of the wide variety of treatment technic (x ray radium surgery) recommended at times somewhat categorically it may be well to consider in a

generic fashion the management of epidermoid carcinoma before undertaking the description of the treatment of skin cancer in specific situations.* Accordingly the treatment of epidermoid carcinoma will be dealt with in the following manner: first, a brief examination of the various factors which influence the selection of treatment, second, a description of irradiation technic as employed in the treatment of skin cancer third, a consideration of the treatment of cancer in specific locations.

Various Factors Which Influence Selection of Treatment Although different methods of treatment are employed by men of wide experience, apparently with equally good results, it appears that the treatment of choice is frequently governed by various factors inherent in each case. If this be true, the end results should be improved if the optimum procedure of treatment is selected for individual cases. Undoubtedly there are today many unknown factors which probably would greatly influence our judgment in the selection of treatment, however incomplete knowledge should not discourage attempts to individualize management in an effort to give the best possible treatment.

Some of the factors to be considered are

- 1 Site of lesion.
- 2 Extent of lesion.
 - (a) Surface area.
 - (b) Thickness or depth.
 - (c) Infiltration of underlying structures.
 - (d) Metastasis.
- 3 Curability.
- 4 Previous therapy.
- 5 Microscopic appearance.
- 6 Known etiological factor.
- 7 Age of the patient.
- 8 Ability of patient to cooperate.
- 9 Facilities available.
- 10 Cosmetic results.

See section on biology p. 140.

A brief discussion of each follows. It must be understood that the various factors weigh with or against each other and all are interrelated.

Site of the Lesion. Lesions in various situations are not all ideally treated with roentgen rays. Notable for their poor tolerance to high doses of irradiation are the scalp, the skin overlying the sternum, the midline of the back, the axillae, groin, vulva, penis, the skin of the leg, particularly that of the medial aspect which is in close approximation with the tibia, and other regions where the skin lies over bony prominences, such as the dorsum of the hands and feet. Only when lesions in these situations are small and superficial should irradiation be considered as the means of cure.

Important, too, in consideration of the type of irradiation treatment to be employed is the possible effect of inadvertent irradiation on vital structures, such as the possible damage to the orbital structures when lesions about the eye are treated with gamma rays. It can be seen that some consideration must be given to the site of the lesion before a decision on treatment is reached.

Extent of the Lesion. Closely related to the site of a lesion is the extent or degree of involvement. The extent of the lesion refers to (1) surface area (2) thickness or depth, (3) infiltration of underlying structure and (4) metastases.

SURFACE AREA. One quite naturally estimates, then measures accurately the size of the lesion as it appears, grossly. Size however is a relative matter. For example a lesion which might be considered small if situated on the abdomen would be considered relatively large if found in the vicinity of the eye, nose, etc.

Generally wide excision should be the treatment of election in situations where

loss of tissue does not seriously compromise the cosmetic results. In other words, lesions of the trunk are usually best treated surgically if operable, since surgical excision can remove more apparently normal skin and tissue than one ordinarily would choose to irradiate. Theoretically the end results should be better and are better if such wide excision is practical. Most primary lesions of the face are best treated with irradiation, although many smaller lesions in the open areas are as well treated by wide cold knife or electrosurgical excision. It must be emphasized that no lesion should be managed surgically if the area of normal tissue planned for excision is less than that area which would be irradiated to a cancer lethal dose if the lesion were treated by irradiation.

When irradiation is used, it should be understood that every portion of a tumor and a zone of apparently normal skin must receive a carcinocidal dose; however as the surface area to be irradiated is increased, the requisite total air dose drops because of increased scattering and increase in skin and depth dose. Occasionally the periphery of a large ulcerated epidermoid cancer does not respond to a dose which ordinarily would be considered sufficient for the lesion. More often the central portion is the most resistant part of the tumor. In such cases, the residue alone may be treated further while protecting the area which has reacted favorably to the treatment already given. If after such added treatment, the desired response is not observed electrosurgical excision is advised.

THICKNESS OR DEPTH. After judging the surface area of a tumor the eleva-

tion above the surrounding normal skin is measured and the degree of depth in infiltration is estimated. Some writers believe that the depth of a tumor is generally commensurate with its external dimensions. This has not been our experience. In general skin cancers unmodified by treatment fall into three groups: those which tend to grow outwardly and show little invasive qualities (most of these are basal-cell carcinomas); those which tend to invade early and show considerably less outward growth (most of these are squamous-cell carcinomas); those which tend to remain flat and do not invade (these are not common and are almost always basal cell carcinomas or Bowen's disease).

The type of radiation to be used should be such that for the size portal required the depth dose is sufficient to sterilize the bed of a thick or infiltrating tumor. This problem is met in several ways. First the total air dose delivered to the tumor can be increased to assure the required sterilizing depth dose. This is an undesirable method. Second the thickness of a tumor can be decreased by subtotal excision using the bipolar endotherm knife, followed by application of low or moderate voltage x rays. This is a satisfactory method. Third high voltage x rays can be used without preliminary surgical modification of the tumor. In our opinion this is the best method of treatment for infiltrating lesions or bulky tumors elevated considerably over 1 cm. above the surrounding normal skin.

INFILTRATION OF UNDERLYING STRUCTURES Deep fascial cartilage or bone involvement should at once exclude the more caustic x rays of low or medium voltage as a modality of treatment.

In a tumor so far advanced as to have involved the deep fascia and tendons of the hands, x rays should not be used at

all as osteonecrosis invariably follows. In addition it is generally conceded that involvement of such structures increases radioresistance. Cases such as these are to be managed surgically.

Cartilaginous involvement does not necessarily contraindicate the use of high voltage heavily filtered irradiation, for in our experience, involvement of cartilage per se has not been the bête noir some believe it to be (see Figs. 20A, 26B, 20A, 20B, 30A, 39B). In any event, operation can always be performed later if wisely employed preliminary irradiation has been unsuccessful.

Where bone is exposed or invaded the important factor to be considered when determining treatment is the extent of involvement. If relatively limited high voltage irradiation results usually in the sequestration of only a small piece of bone. Healing is protracted but the results are usually satisfactory; however in just such cases surgery offers the best prospect for cure (see Figs. 79 and 80).

METASTASIS Ordinarily metastasis should not of itself indicate conservative treatment to the primary lesion. If the primary lesion or the metastatic lesion itself is hopeless, only then should conservative treatment be employed.

Curability Like many bridge players, some therapists are too apt to overbid their hands by attempting to cure every lesion that comes their way. Clearly a line must be drawn for some far-advanced cases are better served by conservative treatment. Intensive ineffectual treatment to a hopeless lesion results only in adding to the discomfort of an already miserable patient. In lesions so advanced that the possibility of a cure is extremely unlikely some treatment must be given if for no other reason than to keep the patient and his money out of the hands of quacks. For such far advanced cases,

fractionated, high-voltage x ray or teloradium, using small daily increments, will do more good and, what is more important, less harm than any other form of treatment. Once in a great while we may be agreeably surprised in an unusually radiosensitive case to find dramatic improvement following such palliative treatment. Then one is justified to continue his treatment in the hope of achieving a successful end result.

low or moderate voltage irradiation has failed to control a resistant lesion, high voltage rays may be tried, but frequently the wiser choice is electrosurgical excision.

Most recurrent lesions do not respond as well to a second course of irradiation as they do to the first. In general, if a dose ordinarily considered carcinocidal has been applied to a lesion and it later recurs, electrosurgery is indicated. On



Fig. 79 Squamous-Cell Carcinoma (Grade IV) Left Involving outer canthus in female aged seventy-five. Right Appearance after 850 rmg. hours of radium and 2900 of high voltage x-ray. Patient developed cataract within one year of treatment. Has been under observation for six years without recurrence.

Previous Therapy The majority of epidermoid carcinomata can be classified as primarily untreated, primarily uncontrolled, and primarily recurrent.

Primarily untreated lesions are managed either by irradiation or surgery, each method dependent on interrelated factors under discussion in this chapter.

Primarily uncontrolled lesions are divided into two groups, those which have received inadequate treatment, those which were resistant. If extremely inadequate previous therapy failed to control a lesion, irradiation still may be tried. If, on the other hand, adequate

the other hand, if an unusually small total dose were given or if the lesion behaved in an unusually sensitive manner high voltage fractionated x rays could be used. A lesion recurrent after adequate high voltage irradiation ordinarily should be excised. It can be seen that in a primarily uncontrolled or a recurrent case, it is essential to have at hand, if at all possible, the full data concerning previous treatment. This is occasionally of great help in deciding the management of the case, for from correspondence with the original therapist the probable radiosensitivity of the tumor

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tion above the surrounding normal skin is measured and the degree of depth in filtration is estimated. Some writers believe that the depth of a tumor is generally commensurate with its external dimensions. This has not been our experience. In general skin cancers unmodified by treatment fall into three groups: those which tend to grow outwardly and show little invasive qualities (most of these are basal-cell carcinomas); those which tend to invade early and show considerably less outward growth (most of these are squamous-cell carcinomas); those which tend to remain flat and do not invade (these are not common and are almost always basal-cell carcinomas or Bowen's disease).

The type of radiation to be used should be such that for the size portal required the depth dose is sufficient to sterilize the bed of a thick or infiltrating tumor. This problem is met in several ways. First the total air dose delivered to the tumor can be increased to assure the required sterilizing depth dose. This is an undesirable method. Second the thickness of a tumor can be decreased by subtotal excision using the bipolar endotherm knife followed by application of low or moderate voltage x rays. This is a satisfactory method. Third high voltage x rays can be used without preliminary surgical modification of the tumor. In our opinion this is the best method of treatment for infiltrating lesions or bulky tumors elevated considerably over 1 cm. above the surrounding normal skin.

INFILTRATION OF UNDERLYING STRUCTURES. Deep fascial cartilage or bone involvement should at once exclude the more caustic x rays of low or medium voltage as a modality of treatment.

In a tumor so far advanced as to have involved the deep fascia and tendons of the hands, x rays should not be used at

all as osteonecrosis invariably follows. In addition it is generally conceded that involvement of such structures increases radioresistance. Cases such as these are to be managed surgically.

Cartilaginous involvement does not necessarily contraindicate the use of high voltage heavily filtered irradiation, for in our experience involvement of cartilage per se has not been the bête noir some believe it to be (see Figs. 28A 28B 29A 29B 30A 30B). In any event, operation can always be performed later if wisely employed preliminary irradiation has been unsuccessful.

Where bone is exposed or invaded, the important factor to be considered when determining treatment is the extent of involvement. If relatively limited high voltage irradiation results usually in the sequestration of only a small piece of bone. Healing is protracted but the results are usually satisfactory; however in just such cases surgery offers the best prospect for cure (see Figs. 79 and 80).

METASTASIS. Ordinarily metastasis should not of itself indicate conservative treatment to the primary lesion. If the primary lesion or the metastatic lesion itself is hopeless, only then should conservative treatment be employed.

Curability. Like many bridge players, some therapists are too apt to overbid their hands by attempting to cure every lesion that comes their way. Clearly a line must be drawn for some far advanced cases are better served by conservative treatment. Intensive ineffectual treatment to a hopeless lesion results only in adding to the discomfort of an already miserable patient. In lesions so advanced that the possibility of a cure is extremely unlikely some treatment must be given if for no other reason than to keep the patient and his money out of the hands of quacks. For such far advanced cases,

microscopic grade or theoretic sensitivity should be treated as radically as possible as too little is known about either to do otherwise.

Certain types of epidermoid carcinoma, such as the trichocarcinoma, cystic, basal cell, etc., which may be recognized microscopically are usually quite radioresistant. In these we use irradia-

usually the diagnosis may be made on the history alone.

Known Etiological Factor: Consideration of the known etiological factor is of help in reaching a decision in treatment in some types of cutaneous carcinoma, such as those developing on a base of a previously existing scar burn scar irradiation damaged skin, varicose ulcer

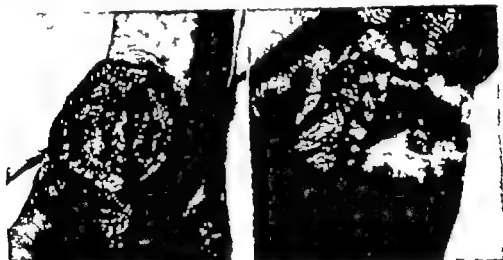


Fig. 81 *Left* Epidermoid Carcinoma (deeply infiltrating, squamous-cell type) in woman aged eighty. Treatment: Midarm amputation, which was easily tolerated under local anesthesia. *Right* Trichosporioblastoma (deeply infiltrating) in woman aged eighty-four after 9600 delivered in three courses, and 116,000 mg. hours (8700 grams) delivered with a 4-gram radium pack. Because of severe pain, patient submitted to midarm amputation, under local anesthesia. Wound healed by primary intention. Patient eventually died of metastasis. If permission could have been obtained, immediate amputation would have been the proper treatment.

tion only in conjunction with surgical excision. Rarely an unsuspected malignant melanoma, which should always be treated by radical surgical excision, is revealed microscopically.

The microscope is of real value when a question of recurrence exists. Trophic irradiation ulceration which may develop belatedly sometimes simulates and should always be distinguished from recurrence. The microscope may also reveal a metastatic skin nodule, but

chronic acrodermatitis, osteomyelitic fistulae, and irritation from arsenic, tar pitch, paraffin, soot, etc. The known sum total of such lesions is low. They are of uncommon occurrence (see Table 11).

Obviously if preventive therapy can be practiced, the possible etiological factors should be eliminated from the patient's environment. Workers should be instructed in methods of protection from irritants peculiar to various types of employment.

can be weighed. It must be remembered that radiotherapy must be effective without doing harm. In recurrent cases the relation between the effective dose and tissue-tolerance dose of the previously irradiated tissues may be such as to invite an unhappy result.

Lesions recurrent after treatment with various caustic pastes are usually less

Basal-cell carcinomas vary widely in their response to irradiation from extreme radiosensitivity to a degree of radioresistance comparable to the average squamous-cell lesion, but as yet one cannot predict the tumor response microscopically. For this reason the only safe dosage for all epidermoid carcinomas is one which may be expected to control



Fig 80: Epidermoid Carcinoma (squamous-cell type Grade III). Left: Deeply infiltrating exposing malar bone in orbiting lid and orbit. Referred to Oncologic Hospital after intensive treatment failed to control lesion. Right: After exenteration of orbit, partial resection of malar bone and wide excision of soft parts. No recurrence fifteen months postoperative.

sensitive to radiation. Such cases are probably best treated by electrosurgery. Lesions which recur after surgical excision can be intensively treated by high voltage irradiation but secondary wide excision frequently is necessary.

Microscopic Appearance. The microscopic appearance will divide epidermoid cancer into three classes: (1) basal-cell carcinoma (2) squamous-cell carcinoma (3) basosquamous-cell carcinoma.

not only the sensitive lesions but also those which are radioresistant. No differentiation should be made between tumor types and all should be treated fundamentally alike, that is, all lesions should receive a required minimal dose; no lesion should receive less. Some basal-cell and many squamous-cell lesions will require more energetic treatment.

On the subject of grading MacCarty states that all cancers, regardless of

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TABLE 11 SITE OF PARAFFIN CRUDE MINERAL OIL, SOOT PITCH, TAR, AND ARSENIC SKIN CANCER

Incident Related to the Cancer	Upper Extremity	Lower Extremity	Scrotum and Perineum	Elsewhere
Paraffin	48	8	45	8
Crude mineral oil	74	22	459	190
Soot	9	0	37	2
Pitch	43	10	148	270
Tar	09	7	108	124
Arsenic	44	20	21	32

Data collected from Harper W C Occupational Tumors and Allied Diseases

The Age of the Patient In reaching decisions concerning management, the patient's structural age is more important than the patient's chronological age. The skin of patients in extreme old age frequently does not recover from radiation effects as well as does the skin of relatively young patients. Moreover in advanced age, ordinarily radiosensitive tumors seem to become less responsive to irradiation. Irradiation should always be protracted and carried to less ascertainable doses. Additional irradiation may be given when more is learned of the patient's tissue tolerance by observation of the effects of the treatment given.

Lesions of the extremity are seen more frequently in the aged. When operation is indicated, elderly patients tolerate surgery surprisingly well, if both cases and operation are wisely selected (see Fig 81).

Ability of Patient to Cooperate Although this should never be a dominant factor in selection of treatment, some consideration should be given to the patient's ability to cooperate in a plan of treatment without undue sacrifice of

time lost from work, travel, etc. It is our belief except in rare instances that the cancer patient should be a lived of the diagnosis. The prognosis to the patient should be good, but he must at least be impressed with the possibility of recurrence. Only in this way can the patient avoid needless worry and appreciate at once the importance of proper treatment and frequent follow up visits.

Facilities Available If after due consideration, the facilities or skill immediately available are not adequate for the needs of a particular case, the patient should be referred to a cancer center able to cope with all phases of the disease.

Cosmetic Results The layman judges the ability of a general surgeon chiefly by the fineness of the scar of the skin wound. The cancer therapist too much concerned with cosmetic results will cure few patients. Desirable as satisfactory cosmetic results may be even in cancer it is a subordinate factor when planning treatment. Radiation will usually produce better cosmetic results if employed when indicated (compare Fig 82 with Fig 83). The same may be said for surgery.

Irradiation Technique in Skin Cancer

First to be considered is the technique employed in the treatment of epidermoid carcinoma in general followed by added pertinent details as they apply to different locations such as the eye, nose, ear, penis, extremities, etc.

Outlined under Radiation Physics are various methods of treatment. The recent trend in the treatment of epidermoid carcinoma has been away from the use of radium, therefore, x-ray technique will receive first and more detailed attention.



Fig. 82: Basal-Cell Carcinoma. *Left:* Right temporal area. *Right:* Appearance after electrosurgical excision and 360 mg. hours of radium plaque therapy.

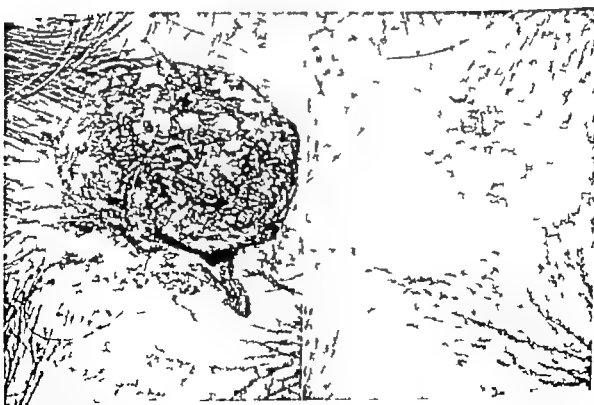


Fig. 83: Basal-Cell Carcinoma. *Left:* Of right temporal area. *Right:* Disappearance after 1800 mg. hours of radium plaque therapy at 2 mm. distance. Treatment given in 8 fractions over a period of one month.

visit, the treatment portal in some cases should be delineated with a nonirritating indelible skin ink. The formula

Cyanolic acid	10.00
Ferric chloride	80.00
Acetone	100.00
Alcohol q. ad	500.00

The mark made by this ink remains visible for several days. On fading, the portal should be redrawn. The aperture through the protecting lead shield, presently to be described, is then shaped and easily fitted to the delineated portal.

Another method of portal delineation is the use of the India ink tattoo dot. Two to three dots suffice to indicate position of shield or mask.

The therapist cannot always include the necessary area of normal skin when thick or infiltrating lesions are treated purely by unfiltered low voltage irradiation. The simple reason for this is that the required total skin dosage of irradiation is almost doubled to obtain the necessary depth dose to the bed of the tumor. The portal size should not be decreased. Either the lesion should be modified surgically before treatment or moderate voltage or high voltage x rays should be used (see under Size of Lesion, p. 165).

Protective Shields. To avoid delivery of undesired radiation to normal tissue beyond the size of the portal planned, some means of defining the field of irradiation is required. Special types of accessory cones are sometimes solely employed but are not to be recommended. Small cones used in the treatment of a small lesion may be properly placed at the onset of treatment, but only a very slight movement on the part of the patient is apt to misdirect the radiation. It is good general practice to employ a cone considerably larger than the planned portal, and to use sheet lead for accurate

delineation of the portal as to size and shape. The sheet lead is $\frac{1}{4}$ inch thick, can be ordinarily quite easily fitted to the anatomical part, and the proper aperture can be easily cut to fit the previously drawn portal. The lead sheet should always be large enough to protect the patient against all undesired



FIG. 84. Master facial lead cast for lesions involving eyelids, nose, or other parts of the face.

direct rays. For lesions involving the eyelids, nose, etc., a rigid lead mask can be swaged to fit a large area of the face (see Figs. 84 to 86). For this purpose, a master facial cast is used. With this technic, rigid portal control is assured. Even though the patient moves slightly, the good sized cone still assures delivery of the radiation to the planned portal.

Protection in the treatment of lesions about the eye will be discussed in detail when treatment of lesions of the eyelids and canthi is considered.

X RAYS

X Ray Apparatus X ray units operated at various kilovolt levels are described in the treatment of superficial cancer. By appreciation of the physical factors involved good results have been reported covering a wide range of kilovolt potentials. It is our opinion that moderate voltage therapy generated at 130 to 140 Kv p is more universally applicable than considerably lower voltages.

Seldom however does a dermatologist possess an x ray unit which can be operated at potentials above 100 Kv p. Although such low voltage apparatus is entirely suitable for many carcinomata of the skin if the physical principles are understood unsatisfactory results attend the use of superficial x rays in any but the most capable hands when large or infiltrating lesions are treated. In addition to serving his patient better the dermatologist will render his specialty an incalculable service by the recognition of the limitations of his equipment and by his refusal to treat any patient who might be treated better by facilities not at his disposal. Because many patients are now seeking treatment earlier in the course of the disease, the majority of patients with primarily untreated lesions who visit the dermatologist can be treated successfully by him.

X RAY FACTORS USED BY AUTHOR

Intermediate Therapy

- 1 140 K. p.
- 2 10 to 15 milliamperes.
- 3 Target skin distance: 21 or 32 cm.
- 4 Filtration equivalent: up to 0.25 mm. of Cu.
- 5 H.V. value layer: 3.8 mm. aluminum with 0.25 mm. Cu.
- 6 Effective wave length: 0.333 Angstrom units.

Superficial Therapy

- 1 100 Kv p.
- 2 5 to 10 milliamperes.
- 3 Target skin distance: 10 to 20 cm.

- 4 Filtration: 1 to 2 mm. of Al.
- 5 Half value layer:
Inherent filter: 0.5 mm. aluminum.
1 mm. aluminum 2.10 mm. aluminum.
- 6 Effective wave length:
Inherent filter: 0.00 Angstrom units.
1 mm. aluminum: 0.425 Angstrom units.

It should be clearly impressed on all that the day of the open bowl apparatus with exposed high tension wires is over and efforts should be made to replace or modify such apparatus.

Treatment Technic *Size of Portal* *Relation to Size of Lesion* A certain width of normal skin should always be included in the treatment portal. Just how much skin beyond the gross limits of the tumor should be treated is not a settled point, but it is generally agreed that too sharp a limitation of the portal regardless of dosage and other factors employed is fraught with danger of recurrence.

A lesion of 1 cm. or less should have a peripheral zone of normal skin 0.5 cm. in width included in the treatment portal as the lesion increases in diameter the peripheral zone of normal skin should accordingly increase so that for lesions 2.5 cm. in diameter the areola of normal skin is no less than 1 cm. in width. For lesions 5 cm. in diameter a width of 1.5 cm. is used and for considerably larger lesions up to 2.5 cm. of normal skin is included (see Chart 2). Some squamous-cell lesions of high grade malignancy may be better treated by including considerably larger areas in the portal for half or more of the treatment. It should be emphasized that these measurements are made from the limits of induration, not visible tumor; therefore, one cannot depend on the technician to perform this duty. In order to make certain that the proper field is irradiated on each treatment

diation for large lesions. Thick lesions considerably larger than 8 cm in diameter are better managed by 200 Kv.p irradiation, using close distance technic that is 30 to 35 cm. T.S.D.

The daily dose is usually 300 r and is carried from 3000 to 4000 r in ten to fourteen days, depending on the response. Immediately following this dosage to the large field, residual areas which have responded slowly are treated while protecting the large field. These more resistant areas are treated through cones or apertures in lead to a total (including previous dose) up to 6000 r or more depending on fractionation.

Area of Normal Skin Irradiated. The normal skin does not tolerate massive doses of unfiltered low voltage irradiation (85 to 100 Kv.p). Since we consider it essential to follow the rules concerning portal size previously given, low voltage irradiation should always be fractionated. We do not favor the use of massive doses delivered to closely delimited fields.

Tk Tube Potential. If, when using unfiltered low voltage irradiation (85 to 100 Kv.p) the lesion is less than 1 cm in thickness, or if a thick lesion has been previously leveled and no evidence of deep infiltration or other contra indications exist (see p. 154) the dose delivered in roentgens falls in the same range as for moderate voltage x rays.

Filtration. It is our opinion that filtered irradiation should almost always be used. For x-rays generated at 85 to 100 Kv.p a filter of at least 1 mm Al is recommended for all except flat lesions. For 140 Kv.p irradiation we use a filtration equivalent of 0.25 mm Cu. With high voltage the filter is usually 0.5 mm. Cu.

Fractionation. Although many therapists employ the massive dose technic,

we believe that fractionation is always desirable. Excluding exceptional cases, fractionation in some degree should be employed to permit recovery of the wide area of normal tissue which should be included in the treatment portal. It should be emphasized that fractionation in no way compromises the end result; the danger of metastasis is not increased and cosmetic results are far better. There is evidence to show that fractionation is always the method of choice. For example Dr. A. Frank, a Viennese radiologist treated a large squamous cell carcinoma in such a manner as to compare the biologic effects of massive and fractional irradiation. He found that the portion of the lesion treated by daily fractionation had completely disappeared, while the side treated with the massive dose still showed viable tumor cells. Others have noted a similar effect. Thus it appears that the use of fractionation in treatment leads to a more rapid disappearance of the tumor than when the massive dose technic is used. This is not a singular experience but is the experience of Hecomet, the author and others.

In the roentgen ray laboratory we not infrequently employ daily fractionated high voltage (200 Kv.p) therapy for fungating and infiltrating lesions and this method of treatment is believed to be distinctly advantageous over other methods in certain cases. This form of treatment is generally regarded as burdensome, tedious, and expensive, yet these should not be considered as deterrent factors in the treatment of any disease particularly carcinoma. The daily increment is generally 300 r. In larger fields and in lesions carried to a reasonable dosage at a rate of 300 r per day where the response indicates an unusually radio-resistant tumor the

The Dose The total dose (see Carcinocidal Dose p 151) required varies according to distance size of lesion area of normal skin irradiated the tube potential filtration the degree of fractionation and the total treatment time for the course

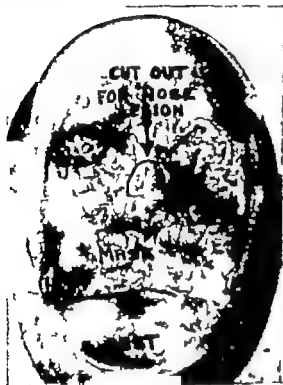


Fig 35 Master facial lead cast.

Distance Excluding so-called contact therapy the target skin distance employed for low or moderate voltage treatment generally varies from 20 to 30 cm. To assure more homogeneous irradiation in large lesions, however the T.S.D. must be increased to 40 cm or more. In exceptionally large lesions, or for large lesions on a convex surface it is better to employ two or more carefully delineated smaller portals rather than a single large one. The smaller fields are desirable because there is a loss of irradiation of from 5 to 30 per cent along the periphery of large convex portals (inverse square law) in addition greater super-

ficial absorption occurs peripherally in bulky lesions due to the oblique entrance of the incident beam peripherally. The T.S.D., when using high voltage irradiation varies from 30 to 50 cm. The percentage depth dose varies directly with the distance (see p 142).

Size of Lesion The size of the lesion per se exerts no influence on the dose for lesions up to 6 cm in diameter using the normal skin margins previously described on p 155. The technique is 500 r delivered every other day to a total minimum dose of 3500 to 4000 r depending on response. Observation two to three weeks later may reveal need of further treatment. If indicated an additional 500 to 1000 r is added. If low voltage unfiltered x-rays (85 to 100 kv p) are used and if the lesion is 1 cm or less in estimated thickness, the same total dose applies. If over 1 cm the lesion preferably should be leveled



Fig 36 Master facial lead cast.

by electrosurgery or the total dosage increased up to 6000 r.

When the carcinoma is more than 6 cm in diameter the total dose is maintained if the lesion is bulky. Except for flat lesions of the intracutaneous type we do not recommend low voltage irra-

mm. of platinum. The lesions are treated at 0.5 cm. to 1 cm. The dose varies from 400 to 600 mg. hrs. per sq. cm., depending on the size of the lesion and the distance, etc.

In this clinic, radium is seldom employed as the primary method of treatment in lesions greater than 3 cm. in diameter or elevated more than half a centimeter and, as a general practice, radium is not used in lesions over 1.5 cm. in diameter. As the dimensions of the portal requiring irradiation increase the assurance of delivering an equal dose everywhere in the treatment field becomes less and less likely. The greatest number of recurrences in reviewing past cases has been in those treated by radium. Warren reports essentially the same experience at the Huntington Memorial Hospital. He attributes the high percentage of recurrences to inadequate screening and insufficient distance. The Radiumhemmet of Stockholm, Sweden, which undoubtedly has had as much experience with radium as any institution, shows approximately the same results as those reported by Warren. Their end results in the treatment of basal-cell carcinoma alone however are excellent.

It should be mentioned that in consideration of the end results of radium therapy a high percentage of treated lesions showed primary healing, only to recur. For example of the forty-four cases treated, forty two healed primarily but recurrences developed in almost 50 per cent (Warren). These failures were due to an inadequate depth dose.

Successfully to employ radium in the treatment of skin cancer demands more than ordinary skill and experience. In addition, it is almost essential that the radium dose be fractionated so as to achieve a greater depth dose as the

tumor shrinks. Further treatment should be applied by a physician rather than a technician and for any patient the same physician should treat the patient throughout. Failing in these recommendations, the end results of radium therapy may be expected to be mediocre at best.

Occasionally the radium bomb is profitably used, but no useful purpose will be served by a discussion of the technique.

Radium and radon used interstitially play an important role in the treatment of metastases and will be discussed in the section dealing with metastases. Interstitial irradiation on rare occasions is useful in the treatment of primary epidermoid carcinoma. Original articles should be consulted.

GENERAL CARE

Proper care of the lesion will permit of larger doses with less damage to the skin. It is good practice to remove the accumulating discharge from an ulcerated lesion at frequent intervals. Larger lesions require hospitalization. The surrounding normal skin is cleansed twice daily with castile soap and frequently anointed with plain yellow petrolatum or olive oil. Occasionally patients are encountered with unusually sensitive skins, at times so rendered by various constitutional diseases medication, etc. In our clinic, the active treatment of syphilis is discontinued for at least one week before and three weeks after irradiation, as the medication employed in the treatment of syphilis (including potassium iodide) increases the sensitivity of the tissues to irradiation. Eczematoid dermatitis is an occasional complication of discharging lesions subjected to irradiation. This complication is usually con-

Fractionally up to 25 per cent.

daily fraction can be reduced in order to extend the period of treatment. Some extremely radio-resistant epidermoid carcinomas apparently respond better to this method of therapy. When moderate voltage or superficial voltage is used 500 r every other day is recommended.

Total Treatment Period The total period of time to deliver a full course (carcinocidal dose) of irradiation for the average cancer of the skin should not ordinarily exceed a period of two to three weeks. The usual period is two weeks. In large fungating radio-resistant lesions, where high voltage daily treatment is used the total treatment period may be as long as four weeks or more depending on the amount of the daily increment and total dose. Judgment based on experience is essential.

Excision Followed by X Ray If curative excisional surgery using either the scalpel or electrosurgery is selected as the method of treatment, the contemplated operation should be planned so as to be curative in itself. There is no reason to believe that inadequate surgery can be salvaged by anything short of a carcinocidal dose of irradiation. In other words, the same irradiation reaction in tissues is probably required to assure sterilization of a few scattered cells as is required for the entire tumor. Furthermore we know that following excision of such lesions including the tumor bed albeit without decisive management the exposed subcutaneous tissue poorly tolerates heavy doses of irradiation. Such conservative excisions, followed by massive doses of from 1200 to 1500 r require six to eight weeks or more to heal. After fractionated doses up to 4000 r (low or moderate voltage) the resultant ulcer requires twelve or more weeks to heal. Such treatment is unjustified. Inadequate surgery followed by

inadequate irradiation adds up to inadequate treatment. Fifty per cent surgery plus 50 per cent irradiation do not add up to 100 per cent cancer treatment! For this reason if conservative surgery is to be used only the fungating portion of the tumor should be removed and a carcinocidal dose given.

RADIUM

Radium Equipment The quantity of radium which should be at hand and the ideal distribution in various applicators are variables which need not be discussed here. The radium distributed in small amounts at the American Oncologic Hospital are in 1 mg, 2 mg, 10-mg, 12.5-mg., 50-mg. and 100-mg. containers, all of which are filtered with 0.5 mm. of platinum with the exception of the 10-mg. and 12.5-mg. needles. These latter filtered with 0.3 mm. of monel metal, are most frequently used in the treatment of skin cancers; they serve multiple purposes.

Technic In the treatment of extremely flat superficial lesions, these caustic monel metal filtered needles are used in direct contact (secondary filter of 1 mm. of rubber). The dose varies from 75 to 150 mg. hrs. per sq. cm. A rather marked reaction follows. The skin surrounding the treatment portal should be protected with 1 mm. of lead thereby allowing the caustic beta rays to act directly only in the treatment portal. This method of treatment is also employed following surgical excision of small but thicker lesions where radiation is desired and the depth dose should be kept at a minimum healing requires four to six weeks.

When other than very flat noninfiltrating lesions are treated the caustic beta rays are eliminated with a filtration equivalent of 1 mm. of lead or 0.5

more severe. The cornea, iris, ciliary bodies, and retina increase in radiosensitivity in the order named.

X Rays—Irradiation Treatment of Chorea Because of the high frequency of cataract and other complications which follow the delivery of inadvertent irradiation to the orbit in the treatment of cancers about the eye the best method of effective irradiation treatment obviously is that which is least likely to deliver unnecessary irradiation to the lens and orbit as a whole. Filtered gamma rays cannot qualify for it is known that a sheet of lead 1 cm in thickness will filter out only about 80 per cent of the gamma rays. Even such an ineffective shield is too cumbersome to be used in practice. When low moderate or even high voltage irradiation is used, from 65 to 99 per cent of the irradiation is prevented from reaching the orbit by a properly fitted eye shield. On the other hand, such a shield filters out less than 5 per cent of gamma rays. Consequently it can be seen that x-irradiation is the treatment of choice for cancer of the eyelid and canthus. Soft beta rays may be employed occasionally as this form of radiant energy can be effectively shielded. The time factor is so short that the gamma ray dose is negligible.

X Irradiation Technique In the treatment of eyelid or canthus lesions, an eye shield should always be used (see Fig 87). Its omission constitutes neglect.

Insertion of Eye Shield The conjunctiva is first anesthetized with two or three drops of 1 or 2 per cent butyn solution. The eye shield (heavily coated with petrolatum) is grasped by the small handle and inserted beneath the lower lid, which is retracted to facilitate introduction. The shield is then introduced beneath the lower lid to such a point

that its upper margin may easily slip under the upper lid. The shield is then placed so as to afford maximum protection and is held in proper position by two or three taut guy sutures anchored by adhesive tape on the forehead and cheek.

Protection of Surrounding Normal Skin After the eye shield has been



FIG. 88 Cystic Basal-Cell Carcinoma. Very early as can be seen. Appearance is similar to that of cysts sometimes seen in this situation. Demonstrates importance of early diagnosis of all lesions by biopsy. Treatment consisted of excision biopsy and roentgen-ray irradiation.

inserted, further shielding of the skin of the face (see pp. 163-164) depends on the type of treatment portal. In the case of a cooperative patient who will not move during the treatment, a proper sized cone may be used without further shielding. This method is not recommended. Movement on the part of the patient when employing such small cones may remove a portion of the treatment portal from the beam, thereby improperly irradiating the cancer. Generally it is better to use a larger cone and a lead shield sufficient to protect the patient. In the absence of cones, the entire head

trolled by employing 0.25 per cent silver nitrate solution as cool wet compresses to the affected part for fifteen minutes four times daily over a period of two or three days

Epidermoid Carcinoma of Eyelids and Canthi

General Tumors of the eyelid are quite common (see Figs 31 to 38). Although papillomata and nevi are seen most frequently about 15 to 20 per cent of tumors of the eyelid are epidermoid carcinomas. In our experience, about one cutaneous carcinoma in eleven is found to involve the lids or canthi. The lower lid inner canthus outer canthus, and upper lid are most commonly affected in the order named. The basal-cell carcinoma is stated to be found more frequently than the squamous-cell type in the ratio of about four to one although in the Oncologic Hospital Series, the ratio is two to one. Twenty five per cent of the lesions measured over 2 cm in large diameter.

Surgery Some carcinomas of the eyelid can be surgically excised with good results, but many more cannot without producing undesirable cosmetic effects. Surgery should be reserved for those cases which fail to respond to irradiation or in cases which have orbital involvement. Surgery of course, plays an occasional important role in the management of metastasis.

IRRADIATION

The Effects of Irradiation Delivered to Region of Eye 1 The possibility of cataract formation when the orbit is unprotected from inadvertent irradiation is a danger which is never to be ignored. Hayes Martin believes its occurrence to be far more common than generally supposed. Driver and Cole

report no cataracts in 324 cases. At least 2 cases were due to irradiation in our series.

2 Corneal ulceration. The cornea is relatively sensitive to irradiation.

3 Permanent epilation of lashes or eyebrows usually occurs following 500 r

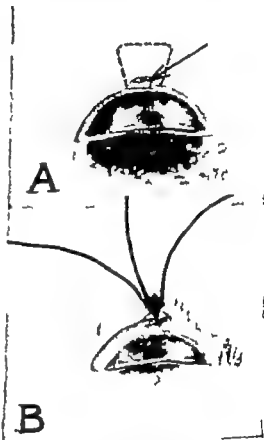


Fig 37 Examples of eye shields.

of low or moderate voltage x ray and always occurs after carcinocidal doses.

4 Contracture with ectropion

5 Stenosis of tear duct. Uncommon

6 Acute glaucoma is occasionally reported due to overirradiation of resistant tumors (without orbital protection)

7 Osteitis of bony orbital rim. Rare complication in relatively small or moderate sized lesions

8 The conjunctiva and eyelids have about the same sensitivity as the skin although the resultant changes may be

recurrence and an inevitable secondary operation, too frequently unsuccessful regardless of its wide extent. If the orbit has become involved its enucleation and a wide excision of the periorbital soft parts is indicated.

a preradiation ophthalmologic examination to determine the status of the lens and other structures of the orbit. Successfully treated cases of inner-canthus lesions may develop epiphora due to a contracture of the puncta, or as



Fig. 90 Squamous-Cell Carcinoma. Left Inner canthus, right eye. Right Appearance six months later. The lesion had received 3900 of high voltage irradiation delivered in thirteen days.



Fig. 91 Basal-Cell Carcinoma. Left Of inner canthus. Right Appearance after 2500 of moderate voltage x-ray

Complications Damage to the tarsal conjunctiva has not complicated intensive treatment in the author's experience. If proper shielding is used and gamma irradiation avoided, few complications need develop. It is advisable to obtain

appears more common, due to a separation of the puncta from the orbital conjunctiva by slight eversion of the lid with resultant loss of the capillary action. For contracture of the puncta, dilation should be postponed for at least

(hair and eyebrows) should be protected with sheet lead 1 mm in thickness, molded and hammered into shape. In this shield an aperture of proper size and shape is made through this aperture the cancer is treated. The lead shields or lead rubber must always be sufficient in size to protect the patient from the direct rays. This cannot be emphasized too strongly!

Various sized cones (always larger than the aperture in the shield) have

lowed by beta and soft gamma ray treatment (see Dose p 148). An eye shield with 1 mm of lead equivalent filters out all the beta rays. The gamma ray dose to the orbit from such treatment (because of the short time factor) is negligible. We have not seen the scarring and retraction claimed by others following such treatment to small lesions.

Recurrent Carcinoma of Eyelid
Following recurrences, careful study should be made of previous dosage. If



Fig. 89: Basal-Cell Carcinoma. *Left:* In region of inner canthus. The dark stain is blood. *Right:* Appearance after radium plaque therapy. No recurrence three years later.

been employed for moderate voltage as well as high voltage treatment to cutaneous carcinoma. This technic has greatly simplified treatment by eliminating the large lead shields.

The Dose The carcinocidal dose for x irradiation is the same as for carcinoma of the skin elsewhere, employing a minimum fractionated total of 3500 r (see pp 152 and 163).

Beta Rays Small lesions of the eyelid not involving the mucocutaneous junction of the palpebral rim can be surgically excised, electrocoagulated and fol-

lowed by high voltage irradiation. If the previous treatment were sufficient in amount, the failure may be due to a too closely restricted treatment portal or an unusually resistant tumor. In the former case the lesion frequently responds to high voltage irradiation, in the latter case, it frequently does not. In any case, high voltage irradiation should always be tried first prior to surgery. If surgical interference is indicated the first operation should be radical in extent. Conservatism results in secondary

recurrence and an inevitable secondary operation too frequently unsuccessful regardless of its wide extent. If the orbit has become involved, its enucleation and a wide excision of the periorbital soft parts is indicated.

a preirradiation ophthalmologic examination to determine the status of the lens and other structures of the orbit. Successfully treated cases of inner-can thus leucoms may develop epiphora due to a contracture of the puncta or as



Fig. 90 Squamous-Cell Carcinoma. Left Lower eyelid, right eye. Right Appearance six months later. The lesion had received 2900 r of high voltage irradiation delivered in thirteen days.



Fig. 91 Basal-Cell Carcinoma. Left Of lower eyelid. Right Appearance after 2500 r (moderate voltage ray)

Complications Damage to the tarsal conjunctiva has not complicated intensive treatment in the author's experience. If proper shielding is used and gamma irradiation avoided, few complications need develop. It is advisable to obtain

appears more common due to a separation of the puncta from the orbital conjunctiva by slight eversion of the lid with resultant loss of the capillary action. For contracture of the puncta, dilation should be postponed for at least

one year. For eversion of the puncta frequent application of olive oil and gentle massage is the best early management. Conjunctivitis which develops from the discharges of the lesion during

important at night. Frequent changes of dressings and irrigations of the eye are essential.

End Results in Treatment of Epidermoid Carcinoma of Eyelids and



Fig. 92. Basal-Cell Carcinoma. *Left:* Of outer canthus. *Right:* Appearance following 3500 r of moderate dosage x-ray. Orbit protected with contact eye shield.

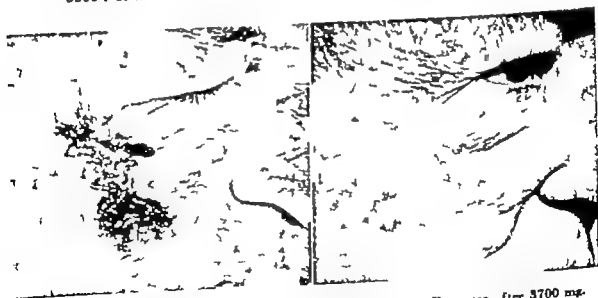


Fig. 93. Basal-Cell Carcinoma. *Left:* Of cheek. *Right:* Response after 3700 mg.-hours of fractionated radium plaque therapy at 0.5 cm. distance and 300 mg.-hours at direct contact. Patient has remained well for seven years.

the radiation reaction and healing period is best managed by prevention. First, the position of the patient should be such as to make the lesion dependent in relation to the orbit. This is particularly

Canthi. In the treatment of cancer of the eyelids and canthi, an analysis of a series of seventy cases* (1930 to 1938) showed that twenty nine (41.4 per cent)

Blipped cases.

had received previous treatment before referral to the Oncologic Hospital.

Uncontrolled Group. In seventeen cases the lesion was never destroyed while under management. In fifteen of these cases information concerning pre-



Fig. 94 Epithelioma. Of eye.

vious treatment was obtainable. Of these fifteen, fourteen had received previous inadequate x-ray, radium, or surgical treatment. All but three were large lesions over 2 cm. in diameter and deeply infiltrating. Six were basal-cell, six squamous-cell, and two basosquamous-cell carcinoma. The patient averaged 69.6 years of age. In two cases the lesions were associated with lupus vulgaris; in one case the lesion was associated with chronic x-ray dermatitis, and one patient was a severe diabetic. The delay interval was known in fourteen patients and was found to average eight years. Those that were followed lived with persistent disease at least an

Interval of time from appearance of lesion to first consultation at Oncologic Hospital.

average of 4.4 years. Nine cases that died of the disease were followed, they lived an average of 2.8 years (including one suicide at one year).

Recurrent Group. Fifty three of the seventy cases in the series showed primary healing after treatment, thirteen of these cases, however, suffered recurrence. An average period of three years elapsed before appearance of recurrence. If one case which recurred after eleven years is excluded the average interval would be 2.1 years. Six of these thirteen recurrent cases were basal-cell, four were squamous-cell, and three were basosquamous-cell carcinoma. Five of the patients had received previous inadequate treatment, four additional cases were over 2 cm. in diameter two were between 1



Fig. 95 Squamous-Cell Carcinoma (low-grade)

and 2 cm. and two were relatively small primary lesions. Five of the recurrent cases had been treated with radium,†

†Doses: (1) 1800 mg. hrs. at 2 mm. distance; (2) 2300 mg. hrs. at 1 cm. distance; (3) 1800 mg. hrs. at D.C.; (4) 500 mg. hrs. at D.C.; (5) 800 mg. hrs. at D.C. The last two were small lesions.

one year. For eversion of the puncta frequent application of olive oil and gentle massage is the best early management. Conjunctivitis which develops from the discharges of the lesion during

important at night. Frequent changes of dressings and irrigations of the eye are essential.

End Results in Treatment of Epidermoid Carcinoma of Eyelids and



Fig. 92: Basal-Cell Carcinoma. *Left:* Of outer canthus. *Right:* Appearance following 3500 r of moderate oblique x-ray. Orbit protected with contact eye shield.



Fig. 93: Basal-Cell Carcinoma. *Left:* Of cheek. *Right:* Response after 3700 mg. hours of fractionated radium plaque therapy at 0.5 cm. distance and 300 mg. hours at direct contact. Patient has remained well for seven years.

the radiation reaction and healing period is best managed by prevention. First, the position of the patient should be such as to make the lesion dependent in relation to the orbit. This is particularly

Canthi. In the treatment of cancer of the eyelids and canthi an analysis of a series of seventy cases* (1930 to 1939) showed that twenty nine (41.4 per cent)

Biopsied cases.



Fig. 96 *Left* Basal-Cell Carcinoma (large) *Center* The defect which resulted after intensive therapy was observed over a period of 1 1/2 months. At the end of that period, no evidence of recurrence was present, and plastic repair was started. The photograph shows forehead (the flap brought down to repair the defect. *Right* Recurrence in graft before completion of plastic surgery two years later.



Fig. 97 Squamous-Cell Carcinoma. *Left* Deeply infiltrating, invading the cartilage of both sides of nose. *Right* After 1837 mg. hours of radium plaque treatment. The lesion recurred three years later. Patient has remained well 6 years after wide surgical excision of recurrence.

including the two relatively small lesions; six others had been treated by a combination of surgery x ray and/or radium one case by x ray alone and one case by surgery alone.

The primary recurrent cases which were followed after secondary treatment (radium and x ray) lived an average of at least two years without further recurrence.

The secondary recurrent cases did not do so well. In none was the disease destroyed but the patients lived an average of six years before finally succumbing to the disease.

It should be added that the end results with high voltage fractionated x rays and radical surgery in the past five years do not present quite so gloomy an outlook for cases previously inadequately treated but the axiom: He who treats first has the best chance to effect a cure still holds true.

Epidermoid Carcinoma of the Nose

The nose is the most frequent single site of cutaneous epidermoid carcinoma (see Figs 96 to 103). One case in four at the Oncologic Hospital involves the nose. The sex incidence is found to be equally divided, the average age, 60.0 years, and the delay interval, 4.1 years. Almost all are of the basal-cell variety although the basosquamous-cell carcinoma is not uncommon (see Table 10). Of eighty lesions studied seventeen involved the tip, twenty the alae or nasolabial fold, eighteen were near the eye-glass area, nine were distributed in the middle third of the bridge and sixteen involved the lateral aspects of the nose. Thirty-three, or 41.2 per cent, of the patients had elsewhere received inadequate treatment by surgery, x rays, radium, solid carbon dioxide, or plastic repair.

Nineteen of the lesions were over 2 cm. in diameter.

In this situation as elsewhere various modalities of treatment are used. Surgery again plays a minor curative role, except in small lesions, because excision of as much apparently normal tissue as is ordinarily included in the portal of irradiation is incapable of attaining cosmetic results comparable to those following irradiation. For small lesions, electrosurgical excision if correctly utilized, is a satisfactory method of treatment. The excision should be planned so as to include as much normal tissue as would be irradiated for a lesion of the same size if this is not feasible excisional surgery is contraindicated. In addition, the incision should extend deeply into the subcutaneous tissues. In small lesions where relatively wide excisions are possible, superficial desiccation or conservative excisions cannot be condoned. We frequently follow these electrosurgical excisions with beta and soft gamma ray treatment delivering about 75 to 150 mg. hrs. per sq. cm. The cosmetic results are good if wound healing is delayed by the frequent application of plain yellow petrolatum. In the past five years we have had not one recurrence following wide electrosurgical excision in primary lesions under 1 cm. in diameter.

If not infiltrative lesions up to 3 cm. in size can be effectively treated by low or moderate voltage irradiation. The dosage factors are the same as those previously described (see pp. 162, 163). Surface radium applicators can also be used for the smaller lesions, except near the canthi (p. 171).

Large lesions over 3 cm. in diameter or those which involve cartilage, are best treated by high voltage (200 K.v.p.) irradiation followed by excision when necessary. Massive lesions involving

surgery for a recurrent lesion should not be considered. Extensive and infiltrating recurrences are best managed by preliminary high voltage fractionated irradiation crossing the nose through lateral portals. Each portal measuring

dependent on the response to the completed treatment general condition of the patient, and other factors.

End Results in the Treatment of Epidermoid Carcinoma of the Nose
Of ninety unselected patients studied



FIG. 100 Squamous-Cell Carcinoma. Left Of nose invading cartilage. Patient was diabetic like female of seventy-nine years. Right Appearance after 4200 r of high voltage fractionated x-ray therapy. Patient has remained well for over eighteen months.



FIG. 101 Basal-Cell Carcinoma. Left Of the deeply infiltrating type. Right Healed after 8400 r fractionated high voltage x-ray therapy. Patient has remained well for over two years.

about 2 to 2½ inches in diameter receives 5000 r (one field treated daily). Following subsidence of the radiation reaction, the further management (additional x-ray or excisional surgery) is

(1930-1938) thirty-one patients or 34 per cent had received previous inadequate treatment before visiting our clinic.

Uncontrolled Group. The neoplasm was uncontrolled in eleven cases or 12.2 per cent. Three of the eleven patients

more than half of the organ are treated by preliminary high voltage irradiation employing two lateral fields with a flat crossfire (eyes protected with lead). The dose is carried to at least 3000 r to each



Fig. 98: Carcinoma.

portal. After subsidence of the irradiation reaction excision of the neoplastic residue is frequently necessary. The extent of the excision varies. Occasionally complete sacrifice of the nose is the only means of cure.

It may be said that small lesions can be effectively dealt with by wide excisional surgery, but can be just as well managed with low or moderate voltage x ray or radium. Moderate sized lesions (uncomplicated) are treated by low or moderate voltage x rays or radium. Large lesions or those with cartilage involvement are best treated by high voltage irradiation and surgery.

In the treatment of cancer of the nose, it must be recognized that there are basal-cell carcinomas as well as squamous-cell lesions which cannot be destroyed safely by irradiation alone. In such stubborn lesions, continued radia-

tion to abnormally high levels unwisely hastens the demise of the patient.

Plastic repair frequently needed, should be delayed for at least one year during which interval a latex prosthesis may be used. Recurrence following extensive plastic surgery frequently eventuates in death (see Fig. 96). Where the original lesion is so extensive that the entire nose is sacrificed the permanent use of a latex prosthesis is ordinarily preferable to rhinoplasty.

Recurrent Carcinoma of the Nose
Factors previously discussed in connection with recurrence apply here (see pp. 184 187 and 190). Conservative surgical or irradiation treatment to small primary recurrent lesions is a frequent and



Fig. 99 Basal-Cell Carcinoma. Of nose. Treated with high voltage x-ray—4500 r delivered in fifteen days. Nineteen days after completion of x-ray treatment, the residue was removed by wide electrosurgical excision. Patient suffered an attack of cerebral hemorrhage thirteen months after treatment, thereby contraindicating planned plastic repair.

inexcusable because of secondary recurrence.

Radium treatment may prove satisfactory but is not as dependable as electrosurgical excision. Immediate plastic

representing 17.7 per cent of the controlled cases, or 18.5 per cent of the entire series. Ten of the fourteen cases had received previous inadequate treatment. The delay interval was known in thirteen of the fourteen cases and averaged 3.8 years, compared with 9.3 years for the uncontrolled series, and 4.1 years for the entire series.

Seven lesions were basal-cell, two were basosquamous-cell, and one was a squamous-cell carcinoma. Two of the lesions were less than 1 cm. in large diameter six were between 1 to 2 cm. and four were over 2 cm. Ten of the cases were treated with surface radium applicators, two by surgery supplemented by radium, one by surgical excision followed by x-ray and one by surgery alone. The average interval before clinical recurrence was found to be 2.04 years. In three cases recurrence took place three years after treatment. One patient suffered three recurrences in nine years. In only two of the cases was it found impossible to control the recurrent lesion. Patients in seven recurrent cases remained well after secondary treatment for periods ranging from two to 21 years, while three patients were followed over one year without secondary recurrence. Excluding salvaged recurrent cases, the total percentage of known failures (primarily and secondarily uncontrolled in our hands) in eighty-seven cases was 11.5 per cent. Fourteen of the patients in the series were lost to follow up or died of other causes before a period of more than two years elapsed. Despite the satisfactory salvage rate in the recurrent cases, the high percentage of recurrence following radium treatment has influenced the change to roentgen-ray treatment in all lesions except relatively flat ones from 1 to 1½ cm. in diameter.

Epidermoid Carcinoma of the Ear

General Carcinoma of the external ear is not a frequently encountered lesion, yet it is not as uncommon as generally believed (see Figs. 104 to 111). One in every eleven cases of cutaneous epidermoid carcinoma seen at the Oncologic Hospital involves the ear.



Fig. 104 Vegetating Epithelioma.
Ear lobes.

Squamous-cell carcinoma involves the ear with greater frequency than basal-cell carcinoma. The ear is the only cutaneous situation of the head where the squamous-cell type predominates. Our series showed 54 per cent of the lesions to be squamous-cell, 35 per cent basal-cell, and 11 per cent basosquamous-cell in type. Twenty-one per cent of the squamous-cell lesions developed metastases.

Men are more prone to the disease than women; in our experience the ratio

Carcinoma of the vermilion border of the lip, strictly speaking, is an oral mucous-membrane lesion.

in whom the disease was not destroyed failed to take treatment. Of the remaining eight cases (all basal-cell carcinomas) six had received previous inadequate treatment elsewhere. The average delay interval was 9.2 years, or more than double the average figure for the series. The average age was 64.7 years.

Six of the lesions were classed as large. Six of the eight patients who were followed until death lived an average of 4.6 years. One of the untreated patients died from an adenocarcinoma of the breast.

Recurrent Group The cancer was apparently controlled in seventy-nine instances but recurred in fourteen rep-



Fig. 102: *Left: Squamous-Cell Carcinoma. Right: Following treatment and repair by Dr. Martin, of Memorial Hospital, New York.*



Fig. 103 *Eptheliomas.*

kerata, produces slow healing ulcers. The tumor bed is essential if such carcinocidal doses are used. Small uncomplicated lesions undoubtedly can be treated effectively with low or moderate voltage x rays without untoward effects, if the tumor bed is left undisturbed. Excision should follow if the response to such treatment is unsatisfactory. When the

though delayed is satisfactory (see Fig 107). If the desired response appears unlikely after carrying the dose to between 4000 and 6000 r at a rate of 300 r per day the patient will usually agree to surgical intervention.

The adoption of high voltage x ray treatment to these lesions has greatly improved the results yet it must be rec-



Fig. 106. Epidermoid Carcinoma. Left: Of ear. Right: Of left buttock.

lesion is small or of moderate size but involves the perichondrium or the cartilage itself, wide excision is undoubtedly the treatment of choice. The average patient, however, rejects the suggestion of an excision and its disfigurement.

If the patient cannot be convinced that surgical interference offers the best and simplest treatment, high voltage irradiation (800 k.v.p.—1 mm. Cu filtration) should be used. Occasionally destruction of cartilage and perforation follow such treatment, but healing, al-

though delayed is satisfactory. Better results will follow if unusually resistant lesions are given the benefits of earlier surgery.

Large lesions of the external ear if operable, are dealt with by amputation.

is 4 to 1 while the experience of others shows a ratio favoring men up to 10 to 1. Patients with carcinoma of the ear are somewhat older than the average patient suffering from epidermoid carcinoma in other sites. The delay interval in men averaged 8.4 years or almost double the average delay interval for epidermoid carcinoma elsewhere.

by excision with the endotherm knife. The base and periphery of the surgical wound are either heavily electrocoagulated or treated with a beta and soft gamma ray surface applicator (see p. 166). The dose is carried to a total of 75 to 150 mg hrs per sq cm (8 to 16 TFD) with the applicator at direct contact. Such treatment is carcinocidal



Fig. 105. Carcinoma. Left: Of ear. Right: Of chin.

Despite a considerable weight of opinion to the contrary, experience indicates that both basal and squamous-cell lesions of the ear do not respond to low or moderate voltage irradiation as favorably as do such lesions situated elsewhere on the face. In nearly 50 per cent of our cases surgery was employed. In many cases, tremendous doses of x and gamma irradiation failed to control the lesion. This poor response to irradiation may find explanation in the relatively avascular thin tumor bed, the presence of cartilage and the sparse stromal tissue.

Treatment. The small lesion which does not involve cartilage is best treated

for a depth of only 2 mm. Healing is somewhat slower than it is following such treatment elsewhere, but the cosmetic results are usually good. A frequently changed yellow petrolatum dressing is used. Some therapists deliver from 500 to 1200 or more r (of variable quality) to the wound following excision. Such treatment is of questionable value for it is likely that the remaining neoplastic cells, if any, will require considerably more treatment before yielding to its effects. It must be recalled that complete excision of cutaneous carcinoma in any situation if followed by x irradiation delivered to carcinocidal

Primary carcinoma of the external auditory meatus is a rare lesion usually recognized at a late stage. Treatment should be high voltage irradiation followed, if possible, by radium inserted into the canal. The radium treatment

tory of previous unsuccessful curative efforts.

Uncontrolled Group. Of fifty cases studied the lesions in thirteen, or 26 per cent, were never destroyed, of the thirteen nine were primarily uncontrolled or recurrent lesions when first seen. Nine of the lesions were squamous-cell and four were basal-cell in type. Ten of the lesions were classed as large and infiltrating. In four cases, the cancer involved almost the entire ear with



Fig. 108 Squamous-Cell Carcinoma. Lesion could not be controlled in male aged seventy.

should be fractionated and protracted over a period of some weeks to avoid a dangerous osteonecrosis. Penicillin therapy appears to be of real value in reducing complications, judging from our recent experience.

In summary small lesions are best managed by excision but can be effectively treated by any type of irradiation. Moderate sized lesions or those involving cartilage ideally are surgical problems, but can be dealt with by high voltage irradiation. Large lesions are best managed by amputation if inoperable, palliative treatment is recommended.

End Results in the Treatment of Epidermoid Carcinoma of the Ear. Of fifty unselected patients studied (1930-1958) 40 per cent related a his-



Fig. 109 Squamous-Cell Carcinoma. This began on the anterior surface of the ear, invaded and broke through the cartilage resulting in globe-like shaped mass with anterior and posterior tumefaction and ulceration. Patient received 6900 high voltage fractionated x-ray in twenty-eight days through 1½-inch cone. The lesion healed in months after start of treatment.

extension to the scalp. Three cases were treated unsuccessfully by radical amputation. Three cases were treated unsuccessfully with high doses (up to 180,000 mg. hrs.) from a 4-gm radium bomb

Generally irradiation offers mere palliation, although an occasional surprising result is encountered in patients who refuse surgical treatment.

The limits of operability are not hard and fast. But the types of cases which do not benefit from radical surgical intervention are (1) large recurrent lesions in which excessive ineffectual irradiation has altered the nutrition of the surrounding tissues with or without asso-

matic response in the unusual case, efforts to effect a cure by irradiation are justified. In such cases the x irradiation treatment is carried to the limits of tissue tolerance followed by interstitial irradiation in the amount of approximately 4 additional T.E.D. (see p 149). The latter treatment is delayed until the x irradiation effects are subsiding.

Lesions situated posteriorly in the sulcus formed by the pinna and the



Fig 107: Epidermoid Carcinoma (squamous-cell type Grade IV). Left: Involving anterior and posterior surfaces of the ear. Treated with high voltage cross-firing fractionated roentgen rays to a total of 4200 r tissue r delivered in twenty-one days through a $1\frac{1}{2}$ inch cone. Right: Lesion healed. No recurrence after three years.

ciated osteitis, (2) large lesions with secondary involvement of the external auditory meatus, (3) large lesions where the neoplastic infiltration extends deeply beyond the limits of the pinna. Although for such cases, palliation is the best that may be offered treatment of some kind (palliative surgery and/or irradiation) should never be refused.

Inoperable primary or recurrent lesions which have spread to involve the contiguous structures of the face or neck are treated by fractionated irradiation. If the therapist is encouraged by a dra-

skin of the mastoid region offer no unusual problem when seen early. Larger lesions in this situation, particularly those with bone involvement, do present a therapeutic problem. High voltage irradiation carried to the usual levels, gradually decreasing the size of the field is usually satisfactory. The residue if any may be cautiously treated further with hard gamma rays. It has been our experience that radiation osteitis is more prone to occur after gamma irradiation than after heavily filtered high voltage (200 Kv p) x irradiation.

Primary carcinoma of the external auditory meatus is a rare lesion usually recognized at a late stage. Treatment should be high voltage irradiation followed, if possible, by radium inserted into the canal. The radium treatment



Fig. 108: Squamous-Cell Carcinoma. Lesion could not be controlled in male aged seventy

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Uncontrolled Group Of fifty cases studied the lesions in thirteen or 26 per cent, were never destroyed, of the thirteen nine were primarily uncontrolled or recurrent lesions when first seen. Nine of the lesions were squamous-cell and four were basal-cell in type. Ten of the lesions were classed as large and infiltrating. In four cases, the cancer involved almost the entire ear with

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In summary small lesions are best managed by excision but can be effectively treated by any type of irradiation. Moderate sized lesions or those involving cartilage ideally are surgical problems, but can be dealt with by high voltage irradiation. Large lesions are best managed by amputation if inoperable, palliative treatment is recommended.

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Fig. 109: Squamous-Cell Carcinoma. This began on the anterior surface of the ear invaded and broke through the cartilage, resulting in globular-shaped mass with anterior and posterior ulceration and ulceration. Patient received 6900 r high voltage, fractionated x-ray in twenty-eight days through a 2½-inch cone. The lesion healed two months after start of treatment.

extension to the scalp. Three cases were treated unsuccessfully by radical amputation. Three cases were treated unsuccessfully with high doses (up to 180,000 mg. hrs.) from a 4-gm radium bomb

Eleven of the patients were followed until death living an average of 2.8 years; six lived only one year or less.

Recurrent Group Of the fifty cases, thirty seven or 74 per cent showed primary healing; eight or 21 per cent recurred. Only two of the recurrent cases had previous inadequate therapy. Six lesions were squamous-cell and only two

were basal-cell. All eight of the cases received inadequate primary treatment judged from present standards. Omitting one questionable case which developed an alleged recurrence after eleven years, the average interval before appearance of recurrence was 2.2 years. One case metastasized three and a half years after successful treatment for a recurrent le-



Fig. 110: Nasal-Cell Carcinoma. Left: Of fossa of right ear. Right: No recurrence three and one-half years after x-ray treatment.

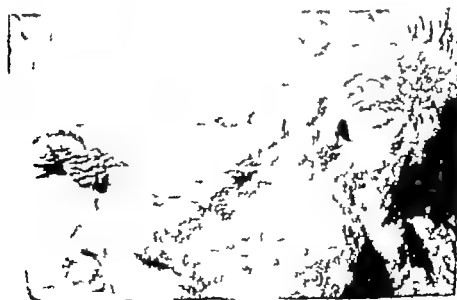


Fig. 111: Squamous-Cell Carcinoma. Appearance following amputation of ear which responded poorly to irradiation. No recurrence eight years after operation.

son Here again is ample proof that skin carcinoma should be followed for a longer period of time than is generally the practice before conclusions are drawn or suggested concerning the possibility of cure

End Results in the Treatment of Epidermoid Carcinoma of the Open Areas* of the Face Of 105 cases studied, nineteen cases, or 18 per cent, received previous inadequate treatment. The average delay interval was 3.8 years for women and 4.8 years for men.

Uncontrolled Group. In 6 of the 105 cases, the cancer was never destroyed while in our care. None of the 6 cases had received previous treatment, but all the lesions were classed as large. All the uncontrolled cancers were of the squamous-cell type and showed an average grade of 3.0 (Broders). The average grade of the entire group was 2.5 while the lesions which healed after treatment showed an average grade of 2.0. Five of the cases were followed until death. The average survival period was found to be 1.4 years—all patients dying directly or indirectly from cancer.

Recurrent Group. Ninety-one of the 105 cases showed primary healing after treatment. Of the 90 healed cases, 8 suffered recurrence. Of the 8 recurrent lesions, 6 were basal-cell carcinomas (2 being cystic basal-cell in type) and 2 were squamous-cell lesions. Only 3 of the lesions were classed as large; that is, over 3 cm. in diameter.

Recurrence was recognized an average of 2.9 years after treatment. Five of the cases suffered secondary recurrence an average of 2.8 years after treatment.

Strangely of the nineteen patients from whom a history of previous inadequate treatment could be obtained, only in three instances did recurrence develop.

**Temple, Clark, et al.*

and in only one case was the cancer never destroyed.

The end results in the treatment of cutaneous cancer of the open areas of the face are far better than are the end results of cancer in other cutaneous situations, as can be seen by simple comparison.

Epidermoid Carcinoma of the Penis

Penile cancer is a relatively uncommon lesion most commonly seen between the ages of fifty and sixty. A biopsy should always be done in order to exclude granulomatous disease which occasionally simulates cancer. It must be emphasized that a specific therapeutic test for syphilis is never to be recommended as a preliminary effort to exclude cancer.

Practically all penile cancers can be classed as radioresistant lesions which require destructive doses of irradiation. For this reason it is apparent that only small superficial tumors are suitable for irradiation treatment (see Figs. 112 and 113). Such lesions, 2 cm. or less in diameter are uncommon and probably make up less than 10 per cent of all penile cancers seen. Lesions of this type with careful shielding can be treated with low voltage or moderate voltage irradiation, using the technique previously described. Surface radium applicators have also been used successfully. It should be borne in mind that the penis does not tolerate irradiation well.

Lesions larger than 2 cm. (see Figs. 114 and 115) are surgical problems inasmuch as they are controlled by irradiation, without serious complications, only in rare and exceptional instances. The surgical treatment usually consists of conservative penile amputation. The radical amputation of former years is



Fig 112: *Left:* Lesion of the prepuce and glans with infiltration. Patient, after refusing amputation, was treated by a combination of high voltage x-ray and radium therapy. *Right:* No evidence of recurrence ten years later.

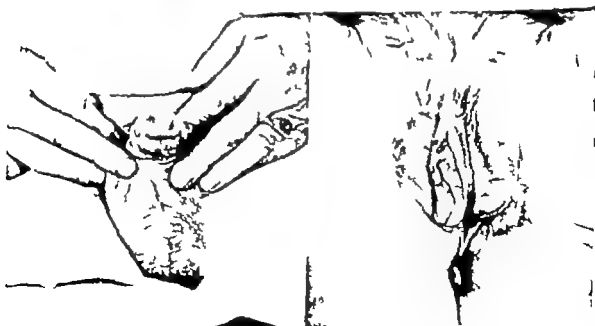


Fig 113: Carcinoma. *Left:* Of penis. *Right:* Of vulva.

ing performed less and less frequently at the present time.

Metastasis develops in about 40 per cent of cases. The poor tolerance to radiation of the inguinal skin, the in-

tastasis is primarily a surgical problem. When palpable metastatic lymph nodes are present and operable, a bilateral radical groin dissection should always be performed. In the absence of palpable



Fig. 114 Left Carcinoma. Right Squamous-Cell Carcinoma.



Fig. 115 Carcinoma. Of testes and scrotum.

ability to employ a crossfire technic, and the radioresistance of the metastatic lesion make it impossible to approach delivery of a carcinocidal dose to this region. Prophylactic irradiation is of no value. It can be seen that me-

metastatic lymph nodes, a prophylactic bilateral radical groin dissection is the ideal procedure. In both instances, the groin dissection should be postponed from three to four weeks after the primary operation.



Fig. 112: *Left: Lesion of the prepuce and glans, with infiltration. Patient, after refusing amputation, was treated by a combination of high voltage x-ray and radium therapy. Right: No evidence of recurrence ten years later.*

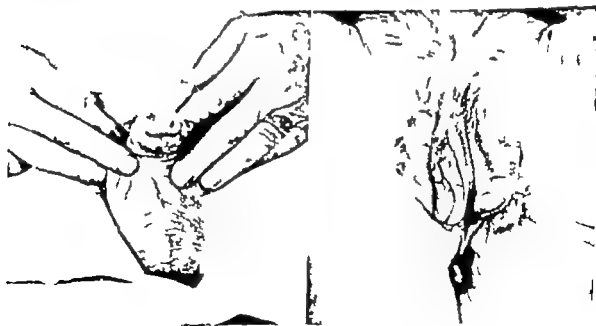


Fig. 113 Carcinoma. *Left Of penis. Right Of vulva.*



Fig. 117 Basal-Cell Carcinoma. *Left* Recurrent, of scalp, after previous unknown amount of x-ray treatment. *Right* After wide excision and sliding flap graft. (Plastic Surgery Service of G. M. Dorrance)



Fig. 118 *Left* After excision of large sarcoma tumor previously inadequately treated with roentgen-ray irradiation. *A* Large sliding flap graft covering defect resulting from excision of tumor and subcutaneous tissue including perianthum. *B* Thiersch graft covering defect of donor site. *Right* After complete healing. Patient has been under observation for two years without recurrence. (Plastic Surgery Service of G. M. Dorrance.)

Inoperable primary lesions and groin metastases may be managed by a combination of palliative surgery and irradiation

Epidermoid Carcinoma of the Vulva

Carcinoma of the vulva is a most serious form of cancer highly disposed to metastasize to lymph nodes regardless of the size of the primary lesion. With this fact all therapists should be familiar.

The lesion is uncommon being only 2 per cent as frequent as carcinoma of the cervix (Tausig). As a rule vulvar carcinoma is found in women past the menopause. Tausig believes that over two-thirds of the cases develop on a basis of generalized or localized leukoplakic vulvitis. He advises prophylactic surgical excision of all leukoplakic vulvar skin.

Authoritative opinion aligns itself almost unanimously against the use of irradiation in the treatment of operable squamous-cell carcinoma of the vulva. This conformity of negative opinion is understandable in consideration of the results obtained by radiation. The percentage of five-year cures achieved by irradiation treatment alone is only 11.0. These palpably poor results from irradiation are ascribed to the marked radio resistance of the squamous cell carcinoma coupled with the marked intolerance of the vulvar tissue to carcinocidal doses of irradiation. Indeed excruciatingly painful irradiation ulcers frequently result and often without destruction of the tumor.

The only indications for radiation treatment are the presence of the lesion in a poor operative risk, such as a feeble old woman, inoperability because of the extent of the lesion, involvement of

the urethra. In the latter case the neoplastic involvement is removed surgically down to the urethra, which is left intact and treated by radium. In the former cases, high voltage filtered x irradiation is the treatment of choice. It is preferable to lightly filtered x rays of low or intermediate voltage.



Fig. 116 Endothelioma Capitis. Lesions are limited to head and face. Duration, 15 years. Size varies from pinhead to walnut and color is brownish-red.

It can be seen that the management of vulvar carcinoma and its regional metastasis must be primarily surgical. The operative technic has become definitely standardized. Five-year end results of radical vulvectomy followed by bilateral groin dissections vary up to Tausig's figure of 64 per cent.

Epidermoid Carcinoma of the Scrotum

Epidermoid carcinoma of the scrotum is usually of the squamous-cell type and in many instances is thought to be asso-

Small noninfiltrating lesions may be managed by x ray or radium. Despite the apparently rich blood supply considerable evidence is mounting to indicate that high doses of irradiation are not well tolerated by the scalp. In addition, osteonecrosis of the underlying calvarium is reported. For these reasons, large, deeply infiltrating, or recurrent lesions should be dealt with by wide surgical excision of all layers of the scalp including the pericranium. The resultant defect may be repaired by a sliding graft (see Figs. 116 and 118). Excision of tissue damaged by heavy irradiation should not be followed by the drilling of burr holes in the calvarium. Such a procedure invites dangerous osteonecrosis and should be condemned.

Epidermoid Carcinoma of the Extremities

General. Epidermoid carcinoma of the extremities is uncommon (see Table 1). It is usually of the squamous-cell type. The delay interval is long and the incidence of metastasis high, probably more than 40 per cent. Notwithstanding the low incidence, these lesions when encountered are important because they are not easily dealt with. The unimpressive end results are probably related to the gravity of these lesions coupled with uncertain fociadative treatment.

The majority of epidermoid cancers of the extremities are found on the hand (see Figs. 119 to 123). Workers exposed to the outdoors, such as farmers, laborers, and carpenters make up the bulk of the cases, and average five to ten years older than patients with cutaneous carcinomas of the face. Frequently seen are associated scale changes, such as keratosis, hyperpigmentation, and atrophy. Radiation dermatitis and scars are also occasionally seen.

Epidermoid carcinoma of the leg is rarely related to a previous or existing abnormal change in the skin. Exceptions are burn-scar carcinoma, which is occasionally seen, and the rare malignant degeneration even in varicose or luetic ulcers, and osteomyelitic fistulae.



Fig. 121. Squamous-Cell Carcinoma of leg. Treated by 1000 mami dose wide excision, and blanket skin graft the following day. Eight months later patient returned with large hard nodule in Scarpa triangle, for which radical node dissection was performed.

Epidermoid carcinoma of the foot is seldom encountered.

Treatment of Primary Lesion. As a rule, epidermoid carcinomas of the extremities are not as responsive to irradiation as are face lesions. In addition, the tumor bed is either situated immediately over bone, or the surrounding skin is so modified by atrophy and the peripheral circulation so inadequate, that carcinoidal doses cannot be safely delivered. For these reasons irradiation as a means of cure should be used only for the smallest and most superficial lesions, and even for these small lesions the most

ciated with a background of chronic irritation *e g.*, prolonged exposure to oils and tars, soot (now rare) paraffin arsenic etc. Although most of the cases are slow growing the long duration and large dimensions lead to a high incidence of metastasis.

The treatment for the primary lesion and metastasis is surgical. Radiation is

reserved as a palliative procedure for far-advanced inoperable cases.

Epidermoid Carcinoma of the Scalp

Most epidermoid carcinomata of the scalp are of the basal-cell type. The uncommon squamous-cell lesions are apparently slow to metastasize.



Fig. 119: Squamous-Cell Carcinoma. *Left:* Of hand, with massive epitrochlear metastasis, in a male aged eighty-two. *Center:* Appearance four months after 3000 r of high voltage x-ray delivered in eleven days. *Right:* Appearance of elbow region after 4200 r of high voltage x-ray was delivered to each of two fields, cross-firing an inoperable met + the mass. Considerable tumor tissue remained after treatment. Patient had refused amputation.

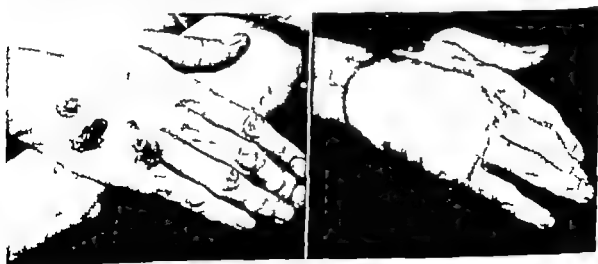


Fig. 120: *Left:* Roentgen dermatitis, keratosis, and microscopically proved carcinoma degeneration. *Right:* After full thickness free-end flap skin graft. The donor site was the abdomen.

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effective and least troublesome method is surgical excision.

When the lesion is deeply infiltrating radical surgical procedures are indicated. In younger patients where the loss of a member for instance the hand is regarded as a serious problem partial hand amputations may be done. In the aged midarm amputations are far more sim-

regional node dissections performed, regardless of the gross status of the lymphatic drainage basin. From a practical standpoint, however we believe that the need for prophylactic lymph-node dissection is variable. For example if the primary lesion involves the extremity of an elderly person and is slow growing, of small size and of a low grade of



Fig. 122 Epidermoid Carcinoma (squamous-cell). *Left:* Large carcinoma involving the right side of the abdomen and groin, which developed in an old burn scar of a female aged fifty. Appearance after patient received 3000 r to a field measuring 15 x 18 cm., and an additional 2400 r to areas of gross residue. Following irradiation, wide surgical excision was performed. *Right:* Appearance after 300 buried pinch grafts were placed in unhealed granulating area, one year after excision.

ple and much more easily tolerated than forearm or hand amputations. In some cases of far advanced lesions of the hand or foot with inoperable metastasis, palliative amputation to relieve distress is justified.

Treatment of Lymph-Node Metastasis. The treatment is primarily surgical. Irradiation is reserved for inoperable and hopeless cases.

Ideally all patients with metastasizing tumors of extremities should have

malignancy a policy of frequent and careful follow up for a period of three to five years is justifiable. On the other hand where such surveillance cannot be anticipated or in cases where the primary lesion is of long duration, or of large size or rapidly growing and of a high grade of malignancy a prophylactic node dissection is urgently indicated.

Visceral metastasis, although not unknown is rare. Generalized visceral carcinomatosis and osseous metastasis from

epidermoid carcinoma of the extremities have been seen. The treatment for such cases is palliative irradiation.

Irradiation finds its place in the conservative management of inoperable or hopeless cases. For example a patient



Fig. 123 Epithelioma. On congenitally deformed hand.

may return with inoperable epistichial axillary or even supraclavicular metastases one, two or more years after successful management of the local lesion. In selected inoperable cases, efforts should be made to control the metastasis by intensive external and interstitial irradiation. In hopeless cases, irradiation should be purely palliative. Irradiation may also be used postoperatively but healing following radical node dissections is frequently unsatisfactory and irradiation, consequently is not well tolerated. It becomes evident that irradiation plays a subsidiary rôle in the management of epidermoid carcinoma of the extremities.

Epidermoid Carcinoma of the Trunk

Epidermoid carcinoma involving the skin of the trunk is uncommon (see Table 1). Most of the lesions are of the flat basal-cell type, Bowen's disease etc. although occasional slow-growing squamous-cell carcinomas usually of large dimension, are encountered. When feasible surgical excision is recommended. In large infected ulcerating lesions, preoperative irradiation has been employed. Wide excision is followed later by placing multiple buried punch grafts in the developing granulation tissue.

Metastasis from squamous-cell carcinoma is not common in small lesions, but its incidence increases sharply in cases where the size of the primary lesion is large and the delay interval is long. The management of metastasis is that



Fig. 124 Carcinoma. Of breast.

described for lesions of the extremities if the lesion is not near the midline and is definitely in the upper or lower trunk. Lesions situated near the midline may metastasize to either side. Likewise such

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Fig. 122: Epidermoid Carcinoma (squamous-cell). *Left:* Large carcinoma involving the right side of the abdomen and groin which developed in an old burn scar of a female aged fifty five. Appearance after patient received 3000 r to a field measuring 15 x 18 cm., and an additional 2400 r to areas of gross residue. Following irradiation, wide surgical excision was performed. *Right:* Appearance after 300 buried pinch grafts were placed in untreated granulating area, one year after excision.

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lesions occupying positions more or less equidistant from the groin and axilla may metastasize to either or both. Because the site of possible metastasis from tumors in such latter situations is unpredictable, prophylactic node dissections should not be performed.

Epidermoid Carcinoma of the Lip

Strictly speaking epidermoid carcinoma of the lip is not a cutaneous lesion

years (average) after the trauma. The prognosis is less favorable than in the acute type. All chronic burn-scar cancers are best dealt with by excision, as resistance to irradiation is the rule. Large lesions, however, are sometimes intensively irradiated before excision. Amputation is advised for large lesions of the extremities; all those which show deep infiltration call for amputation. The best treatment is preventive—that is, promo-



Fig. 125 Epithelioma. Left: Before treatment. Right: After treatment.

but an intraoral mucous membrane tumor. A full understanding of the problems involved is essential. Irradiation and surgery have unique advantages in the management of the primary lesion and metastasis.

Skin Cancers Associated with Definite Stimulating Factors

Burn Scar Cancers. The acute type may appear from one to two years after a relatively superficial small burn. In all respects it behaves as does the ordinary spontaneous cutaneous carcinoma and treatment is selected dependent on other factors (see p. 154).

The chronic type appears about thirty

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Scar Cancers (Marjolin's Ulcers). These are slow growing and slow to metastasize. Few are basal-cell in type. Although occasional cases with short latency periods (six months to three years) are reported, in the majority of cases the latency period is prolonged from twenty to forty years (Hueper). These cancers are best managed surgically because of the dense and avascular fibrous tissue tumor bed.

Cancer Developing in Lupus Erythematosus and Lupus Vulgaris. These cancers are essentially scar cancers. Management is surgical.



Fig. 126 Adenocystic Basal Cell Epithelioma. *Left* Of forehead in white woman aged sixty-six. *Right* Same case after sliding scalp flat. Patient died of carcinoma of the rectum three years later (Plastic Surgery Service of G. H. Derrance)



Fig. 127 Epithelioma. *Left* Of lower lip, before treatment. *Right* After treatment.

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Fig. 129 *Left* Prickle-Cell Carcinoma. Of Hp. *Right* Carcinoma. Of tongue



Fig. 130 Carcinoma.

Cancer Developing in Varicose Ulcerations and Chronic Acrodermatitis Atrophicans. Squamous-cell carcinoma uncommonly complicates varicose ulceration and chronic acrodermatitis. The duration of ulceration preceding malignant degeneration is said to average about twenty years. Treatment presents



Fig. 128. Squamous-Cell Carcinoma. Appearance 1 1/2 months before referral to Oncologic Hospital. Not radiation-damaged skin and squamous-cell carcinoma of skin of upper lip which developed following degeneration of trophic ulcer due to excessive unfiltered superficial irradiation employed in the treatment of eczema 14 years before.

a serious problem since the tissues in each condition heal poorly following any form of management. Irradiation is especially poorly tolerated. Wide surgical excision and immediate skin grafting is the procedure of choice. Metastasis to lymph nodes is slow to occur because of the obliterated lymphatics, but metastasis via the blood stream is reported (Pack).

Cancer in Osteomyelitic Fistulae. Epidermoid carcinoma may rarely arise in and around chronic osteomyelitic fis-

tulae. The latency period varies from twenty to fifty years. Severe pain and increased amount of discharge are indications of malignant change. The cancer displays a tendency to remain localized for a long period, offering therefore a favorable prognosis if local treatment is adequate. The lesion in the great majority of cases develops in osteomyelitic fistulae of the lower extremity (Hueper). Amputation generally is the treatment of choice.

Xeroderma Pigmentosum. This condition is apparently sensitive to any form of radiant energy and irradiation therapy is therefore contraindicated. The keratoses and cancers which develop should preferably be excised or electrocoagulated as they appear.

Roentgen or Radium Cancer. The majority of roentgen or radium cancers are cornifying squamous-cell carcinomas which develop on a basis of chronic radio-dermatitis. The latency period averages seven to nine years or more (Hueper). About 25 per cent of cases show metastasis which in general is delayed but can be early and fulminating.

The use of roentgen rays or radium in the treatment of operable irradiation cancers is contraindicated. Only in inoperable or hopeless cases should irradiation be employed where beneficial palliative values occasionally may be obtained. Severe and fatal recurrence generally follows irradiation treatment (Ullmann).

Arsenic, Tar Pitch, Paraffin, Crude Mineral Oil, and Soot Cancers. These lesions are now of low incidence at least in this country due to the employment of preventive measures in industry. Medicinal arsenic cancers are still encountered occasionally. Exposure time for this group of lesions varies from fifteen to thirty years or more before cancer develops. Arsenic cancers generally ap-



Fig. 129 Left Prickle-Cell Carcinoma. Of lip. Right Carcinoma. Of tongue



Fig. 130 Carcinoma.

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pear earlier (one to ten years). This group of cancers is squamous-cell in type, generally slow growing, and slow to metastasize. The majority of these lesions involve the scrotum (see Table 11) in this situation, they are best managed surgically. Elsewhere, radiation can be employed if the cancer is not of large size.

Lymph-Node Metastasis

Prophylactic Irradiation The designation prophylactic irradiation is generally applied to treatment delivered to a lymphatic drainage basin in the absence of clinically demonstrable nodes, the purpose being to destroy microscopic metastasis which may possibly exist or to prevent or make less likely the development of future metastasis in the treated area.

Obviously any ultimate value for prophylactic irradiation could actually exist only for those cases which would develop regional metastasis after successful treatment of the primary lesion. The hypothetical value of prophylactic treatment is almost nil if judged by the percentage of cases developing node metastasis after the primary lesion is successfully treated. When free of metastasis on admission less than 2 per cent of cases of squamous-cell carcinoma of the face ever develop regional node metastasis, if the primary lesion were successfully treated. Therefore in 98 cases out of 100 the prophylactic treatment would not have even a hypothetical value.

Consider now the actual value of prophylactic irradiation in the two cases out of 100 which may be expected to develop metastasis after a primary cure. Little or no evidence exists to show and few radiologists believe, that impalpable microscopic metastasis can be sterilized by dosage as ordinarily employed in pro-

phylactic irradiation (2 to 3 T.E.D.) In consideration of higher doses, it is generally agreed that efforts to administer carcinocidal doses to an entire lymphatic drainage basin as a routine purely prophylactic measure are unjustified in consideration of the very low incidence of metastasis following successful treatment of primary epidermoid carcinomas of the face. Prophylactic irradiation is also employed by those who entertain the hope that the lymph node bearing area shall thereby be rendered more resistant to metastatic spread even though no actual carcinocidal value exists. There is little clinical or experimental justification for such hope. Prophylactic irradiation no longer may be regarded as a recommended procedure when dealing with epidermal carcinoma of the face.

Arguments could be presented to show that prophylactic irradiation is not only useless, but actually harmful, when employed for cases of epidermal carcinoma of the extremities. Only rarely do indications and circumstances exist which would make such treatment an acceptable method of management.

Prophylactic Dissection of Regional Nodes Prophylactic dissection is not employed for squamous-cell carcinoma of the face. In situations where the incidence of metastasis is high, such as the vulva, scrotum, and penis, a prophylactic dissection is indicated (see p 192).

Small lesions of the scrotum or penis do not require prophylactic dissection if a careful follow-up is maintained. Metastasis from lesions of the extremities is discussed on p 192.

Possible Sites of Metastasis for Epidermoid Carcinoma of the Skin The therapist who is not familiar with the possible routes of metastasis from lesions of different situations does his patient an injustice, since a follow-up



Fig. 131: *Left:* Pedicle graft sutured to defect. Skin to be substituted for mucous membrane. *Right:* End result. No recurrence in 11 months and one-half years. (From Plastic Surgery Service of G. M. Dorrance.)



Fig. 132: Squamous-Cell Carcinoma. *Left:* Defect following electro-surgical excision of recurrent carcinoma of skin of upper lip, previously inadequately treated elsewhere. *Right:* Pedicle graft the base of which has been freed and sutured in original position.

TABLE 12. EXPOSURES NECESSARY TO DELIVER SPECIFIED MINIMUM DOSES IN VARIOUS TISSUE VOLUMES

Interstitial Sources Filter 0.5-0.5 Mm. Pt

T.E.D.	Diameter of Spherical M. in—Cm.									
	1.0	1.5	2.0	2.5	3.0	4.0	5.0	6.0	7.0	8.0
	1 Liter—Cubic Cm									
	9.5	18	40	80	130	210	350	1150	1900	2750
T.E.D.	Mg. or M. Hour to Deliver Specified Dose									
	1	2	3	4	5	6	7	8	9	10
1	33	90	133	230	330	480	600	673	800	1150
2	70	180	270	500	660	900	1000	1150	1300	1550
3	103	270	403	730	960	1300	1500	1700	1900	2250
4	140	360	540	1000	1340	1800	2000	2300	2600	3000
5	175	450	675	1230	1630	2200	2400	2700	3000	3500
7	243	600	913	1700	2250	3000	3300	3700	4200	4800
10	330	800	1230	2300	3000	4000	4500	5000	5600	6400
T.E.D.	Millimeters Diameter to Deliver Specified Dose									
	1	2	3	4	5	6	7	8	9	10
1	0.65	0.6	1.0	1.5	2.5	3.5	5.0	6.5	8.5	11.5
2	0.80	1.2	1.5	2.5	3.5	5.0	7.0	9.0	12.0	15.5
3	0.75	1.8	2.0	3.5	5.0	7.0	10.0	13.0	16.0	20.0
4	1.00	2.5	3.0	5.0	7.0	10.0	13.0	16.0	20.0	25.0
5	1.25	3.0	3.5	6.0	8.0	11.0	14.0	17.0	21.0	26.0
7	1.75	4.0	4.5	7.5	10.0	13.0	16.0	20.0	24.0	30.0
10	2.50	6.0	7.0	10.0	13.0	16.0	20.0	24.0	30.0	36.0

Permission of E. H. Quinlan; Courtesy of Edwards Brothers

12 T.E.D. or more for its control. Such dosage cannot safely be delivered to any great volume of tissue regardless of the means employed. High voltage (200 Kv.p.) fractionated irradiation is used. Doses range from 3000 to 6000 r delivered at a rate of 300 r per day through a filter of 2 mm of copper at 50 cm T.S.D. Circular fields about $\frac{1}{2}$ cm greater than the node are used if clinical evidence indicates that the metastatic disease has not extended beyond the thick capsule of the cervical node. Occasionally by careful technique cross-firing is feasible thus making possible

the delivery of sterilizing doses by external irradiation alone in rare instances. If, as occasionally happens, several separated metastatic nodes are present, they should be treated individually rather than by the employment of a blanket field.

After completion of the course of external irradiation, the tumor dose is estimated by reference to proper depth dosage tables. Before subsidence of the radiation reaction and generally immediately after completion of the external irradiation supplementary interstitial irradiation is employed in the form of

examination must exclude the development of metastasis in the sites of predilection. Too frequently it is the patient who calls the attention of the physician to a metastatic node. It is, therefore, essential for the physician to have in mind the possible sites of metastasis, the more important of which are

Scalp lesions

Posterior aspect	Occipital nodes
Parietal aspect	Mastoid nodes
Frontal aspect	Preauricular nodes

Eyelids

Posterior aspect	Mastoid nodes
Anterior aspect	Preauricular nodes, subparotid nodes, sublingual nodes, infraorbital nodes

Face

Middle of face (extending in line of lower lip)	Mandibular nodes, posterior, inframaxillary nodes
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Skin of upper lip	Crossed metastases
Lateral aspects of face	Preauricular nodes, subparotid nodes, sublingual nodes

Trunk lesions

Lower trunk	
Superficial	Superficial femoral, deep femoral

Infiltrating	Superficial femoral, deep femoral, external iliac nodes
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Upper trunk	Villary nodes, cervical nodes, internal mammary nodes
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Middle section	Either side or bilateral
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Perineal and rectovaginal lesions

Superficial femoral, external iliac glands; crossed and bilateral metastasis occurs

Lower extremity

Posterior	Popliteal nodes, deep femoral nodes
Anterior	Superficial femoral, iliac nodes (late)
Upper extremity	Epitrochlear nodes, axillary nodes, supraclavicular nodes (late)

The Management of Lymph Node Metastasis The method of treatment for metastasis is determined by the status of the primary lesion, the situation of the primary lesion and metastasis, the local operability of the metastasis, the age and general condition of the patient, etc.

It must be stressed that the primary cancer must be curable and adequately treated before radical surgical intervention is considered for the metastasis. If the local lesion is regarded as hopeless, the metastatic disease regardless of its extent should be treated with palliative doses of irradiation.

Metastatic disease from local lesions involving the extremities and trunk are managed surgically when operable. Supraclavicular metastases from cancers of the upper extremity are generally regarded as inoperable. In such cases, the axillary metastasis, usually present, and the supraclavicular metastasis are to be treated by irradiation.

Metastasis developing from cutaneous carcinoma of the head may be managed surgically if operable but irradiation is demonstrating its ability to cope with many of these lesions. Metastasis from squamous-cell carcinoma of the skin of the head is rarely fulminating and is frequently to be found in the preauricular region or closely related to the parotid gland and facial nerve. In such cases, radiation is not only justifiable but probably will become the recommended procedure.

Radiation therapy is successful only when the metastasis is confined to a relatively small area. External irradiation supplemented by interstitial radon or radium is the usual procedure. It is well known that metastatic squamous cell carcinoma is generally quite resistant to irradiation requiring probably 7

radium needles, or preferably gold or platinum radon seeds. The aim of such additional treatment is to bring the tumor dose into the carcinocidal range without irreparably damaging the integumental tissue. As previously stated, probably from 7 to 12 T.E.D. (into the tumor) are required to destroy these metastatic foci. The dose required from the interstitial irradiation is dependent upon the dose delivered by preliminary external irradiation, and the response of the lesion to the preliminary irradiation. The sum tumor-dose achieved by external plus interstitial irradiation should be between 7 and 12 T.E.D. The dosage in millicurie destroyed or milligram hours of interstitial irradiation to deliver the required dose in T.E.D. varies with the volume and spatial form of tissue irradiated, intensity of implants, etc. The dosage may be computed by reference to proper tables, an example of which is shown in Table 12.

Melanoma and Other Malignant Skin Tumors

The melanoma is a malignant neoplasm springing from cells which have the capacity of producing the pigment melanin. It is believed by some that the melanoma may spring from either ectodermal or mesodermal cells; in short, that a melanoma may be either a carcinoma or a sarcoma, the diagnosis resting upon the microscopic picture and the first mentioned being far more common. However because of the uncertainty of origin of many melanomas, it is sometimes the practice to avoid subdivision into classes, such as melanocarcinoma or melanosarcoma, by using the generic designation malignant melanoma.

Etiology Probably less than 10 per cent of skin cancers are malignant melanomas. The higher incidence reported

from some cancer clinics is explained by the natural gravitation of grave cases to such centers. In many if not most cases, melanomas develop in previously existing moles. Nearly all melanomas are found externally where no especial site of predilection exists. Chronic trauma to an antecedent mole is thought to be a factor in a considerable percentage of



Fig. 125 Malignant Melanoma. Recurrent, of back, in female aged fifty-seven. Patient succumbed eight months later with auxiliary liver and brain metastases.

cases. In rare cases, the primary site of the disease cannot be shown.

Course Although the lesion may yet be justly regarded as one of the most treacherous neoplasms encountered in oncology the complete despair of former years is lifting. From a beginning when scarcely no one could report a cure, we have today arrived where an occasional case, even with proved regional metastases, survives the five-year period. De Chabauty reports 42.8 per cent five-year cures, and from a survey of the literature he finds a 10 per cent average for five-year cures.



Fig. 133: Malignant Melanoma. *Left:* Between fourth and fifth toes of right foot. Patient received preoperative high voltage therapy to a total of 3600 r. Surgery had been refused. *Right:* No evidence of recurrence or metastasis one year after wide surgical removal. More radical surgery refused.



Fig. 134: Malignant Melanoma. *Left:* Of medial malleolar area, resulting from degeneration of previously existing mole. *Right:* After wide excision and full thickness skin graft.

case. These latter lesions are referred to as melanoma-nidus-melanin or amelanotic melanoma.

Treatment. *Preventive.* Almost any individual may count a dozen or more pigmented moles upon his body. This, and the added fact that of the billions of pigmented nevi extant only an infinitesimally small percentage undergo malignant melanomatous degeneration, make purposeless routine prophylactic excision of these benign lesions. When situated where constant irritation is likely nevi should be removed by complete excision. Either electrosurgical or sharp-knife excision with primary closure is a satisfactory method of treatment. Incomplete electrocoagulation of pigmented moles is to be regarded as a dangerous procedure. When moles are electrocoagulated the operator must be certain that the lesion has been completely destroyed. Preliminary circumcavalation will insure coagulation to a proper depth. Irradiation should never be used to treat pigmented moles as they are extremely radioresistant.

Radiation Therapy. The melanoma should be regarded as an extremely radioresistant lesion. Pack and Laing show that only about 2.5 per cent of lesions disappear after necrotizing doses of irradiation. Irradiation should never be used in the treatment of operable cases. Indeed its value for hopelessly inoperable cases is placebo.

Surgical Cases Without Evident Lymph Node Metastasis. The local lesion should be dealt with radically by surgery. Malignant melanoma of the hand or foot in many cases is best managed by amputation. When situated elsewhere, wide excision of apparently normal skin and wider excision of subcutaneous tissue and underlying fibrous fascia is advised whenever feasible. Some

writers advocate the excision not only of the primary lesion but of a strip of skin and fascia extending from the primary lesion to the regional nodes. Such a procedure is not applicable in most cases.

Because of the high incidence of node involvement, even though not clinically



Fig. 137 Malignant Melanoma.
Of postauricular region.

evident, a radical groin dissection should be done in every case. The operation, however, is not to be performed at the time of the operation for the local lesion. It is best to wait an interval of three or four weeks after healing of the primary operation.

Cases with Operable Regional Metastasis. The management of the local lesion is the same as in the group discussed here, too. The regional node dissection should be delayed until complete healing of the primary operative

Although in many instances the onset and clinical course of the melanoma are inexplicable, the most frequent history is perhaps that of *change* in a previously existing pigmented mole. Metastasis occurs early or late by way of the lymphatics or blood stream. Cases are known to have remained locally cured

noma, knowledge of the structure of the benign pigmented mole is essential. Both ectodermal and mesodermal elements are present in the tactile end organ of the sensory nerve filaments of the skin from which the melanoma is thought to develop (Masson). The microscopic appearance therefore is variable, dependent



Fig. 136: Epidermoid Carcinoma (squamous-cell). *Left:* Appearance before treatment of an ulcerated, deeply infiltrating lesion of two years duration, in a male aged forty-four. *Pathological diagnosis:* malignant melanoma due melanin. *Center:* Same patient after 4100 r of high voltage, daily fractionated therapy. Because of prompt response to treatment, the original diagnosis was questioned. Another biopsy submitted for study was reported epidermoid carcinoma, squamous-cell type, Grade IV. The absence of response to therapy as an aid in diagnosis is illustrated. *Right:* Excellent cosmetic result. No evidence of recurrence 18 months later.

for periods ranging up to twenty years, only to succumb at that late date to remote metastasis. Small lesions with delay intervals of about one year show metastasis in about 50 per cent of the cases. When the delay interval reaches three years, up to 80 per cent of the larger lesions reveal metastasis (Taylor and Nathanson).

Histopathology To understand the histopathological structure of the mela-

upon which of the elements, ectodermal or mesodermal is the actual seat of the neoplastic change. For this reason probably no tumor presents so variable a picture. The appearance may be that of a carcinoma with large cells frequently approximating an alveolar arrangement or it may have the appearance of a sarcoma with an unusually varied cellular pattern. Pigmentation which is usually heavy may be entirely absent in some

cases. These latter lesions are referred to as melanoma-une-melanin, or amelanotic melanoma.

Treatment. Preventive. Almost any individual may count a dozen or more pigmented moles upon his body. This, and the added fact that of the billions of pigmented nevi extant only an infinitesimally small percentage undergo malignant melanomatous degeneration, make purposeless routine prophylactic excision of these benign lesions. When situated where constant irritation is likely nevi should be removed by complete excision. Either electrosurgical or sharp-knife excision with primary closure is a satisfactory method of treatment. Incomplete electrocoagulation of pigmented moles is to be regarded as a dangerous procedure. When moles are electrocoagulated, the operator must be certain that the lesion has been completely destroyed. Preliminary circumcirculation will insure coagulation to a proper depth. Irradiation should never be used to treat pigmented moles as they are extremely radioresistant.

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Because of the high incidence of node involvement even though not clinically



Fig. 137. Malignant Melanoma. Of perianal region.

evident, a radical gross dissection should be done in every case. The operation, however, is not to be performed at the time of the operation for the local lesion. It is best to wait an interval of three or four weeks after healing of the primary operation.

CASES WITH OPERABLE REGIONAL METASTASIS. The management of the local lesion is the same as in the group discussed. Here too the regional node dissection should be delayed until complete healing of the primary operative



Fig. 138: Melanosarcoma. *Left:* Large lesion, of one year duration, involving lateral aspect of thigh. Pathological diagnosis: melanosarcoma. *Right:* Same patient after wide excision by means of electro-surgery. Following granulation of wound, multiple buried pinch graft were placed. Patient has remained well for four years without evidence of recurrence or metastasis.

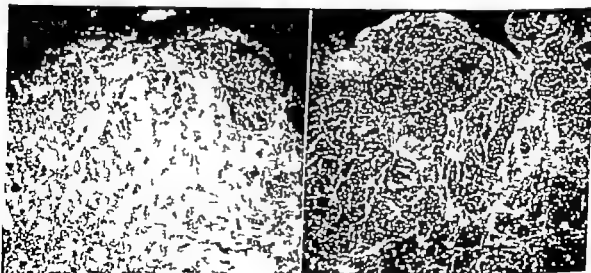


Fig. 139: Malignant Melanoma. *Left:* Of the inguinal skin. The cellular pleomorphism seen in this section is characteristic feature in many melanomas. *Right:* Of the skin of the ankle. The nests and columns of cells immediately beneath the epidermis are nevusoid in type, but the invasion identifies its malignant character. Scattered throughout the section, melanotic pigmentation can be seen.

site. The end results of operative efforts to cure melanomatous lymph-node metastases are most unimpressive, probably less than 15 per cent but occasional cures can be obtained, and it must be emphasized that no alternative method exists which can offer any hope.



FIG. 140 Malignant Melanoma. Patient noted active growth in small pigmented mole situated just anterior to the tragus of the left ear. In one year the malignant melanoma had grown to involve almost half the scalp. Patient was not treated.

CASES WITH INOPERABLE REGIONAL NODE METASTASIS OR DISTANT VISCERAL METASTASIS. Where inoperable regional metastasis exists, simple excisions of the primary lesion are adequate for the need. If the case irradiation may have some slight value in the treatment of the inoperable metastasis. Occasionally conservative surgery is required in the management of the lymph-node metastasis. When distant metastasis exists, seldom is operative interference indicated for the drainage basin, regardless of its status.

Other Malignant Neoplasms of the Skin. Primary sarcoma of the skin is encountered rarely. Sarcomas of connective tissue origin such as fibrosarcoma, myxosarcoma, and liposarcoma are those most frequently seen. Less frequently seen are sarcomas of nerve and muscle origin. Because of the radioresistance of these lesions they are best dealt with by surgery. The prognosis is quite favorable if the lesions are attacked early or are of low grade. Metastasis, which takes place through the blood stream more commonly than through lymphatic channels, makes for a poor prognosis in any



FIG. 141 Recurrent Melanocarcinoma. In white female aged fifty-three. The right axilla, site of the lymph node metastasis, was previously operated on. Primary also never found; metastasis is clearly lymphatic.

but the earliest lesion. Equally as rare as these tumors is the angiosarcoma. It differs from the above group in that it is radiosensitive and best treated by radiation.



Fig 142 Melanoma. *Left:* Lesion of three years duration. Biopsy reported as melanocarcinoma size m l nln. *Right:* Appearance after radical operation which included wld skin excision, removal of pectoral muscles, and axillary dissection.



Fig 143 Melanotic Sarcoma

Lymphomatous tumors occasionally develop markedly radiosensitive skin infiltrations. Malignant neoplasms of almost any origin may on occasion metastasize to the skin (see pp 157 and 207). Treatment is dependent on many factors; they cannot be enumerated here. It can be stated, however, that intensive therapy is never employed; palliation only is indicated.

Indications for Surgery in Skin Cancer

A detailed description of surgical technique as used in the management of cancer of the skin is beyond the scope of this work. Except for minor procedures such as biopsy fulguration, etc., a thoroughly adequate oncologic surgical background is expected of the operator. Inasmuch as this is the case the needs of this text are probably best served by an enumeration of some of the various uses for surgery as it pertains to skin cancer. In some cases the surgical indications may be regarded as absolute; in a narrowing middle group of cases, the question of whether surgery is absolutely indicated is unsettled because of recent advances in high-voltage irradiation therapy; in other cases, surgery is admittedly a purely elective procedure. In other words, some cases demand surgery; others do not, and a group exists in which there is a difference of authoritative opinion.

Today it is generally conceded that radiation is usually the primary treatment of choice for lesions about the head and neck. Elsewhere, surgery generally is to be preferred. It must be emphasized that a lack of understanding of the tenets of treatment, using either irradiation or surgery, will be productive of poor results. Each method is finding its place.

Indications for Surgery

Absol. (surgery essential):

- Malignant melanomas
- Carcinoma of the alvea
- Carcinoma of the penis
- Carcinoma of the cervix and groin
- Regional lymph-node metastasis from epidermoid carcinoma of the extremities
- Bulky tumors (modification of lesion prior to irradiation with low-voltage x-rays)
- Recurrence following previous adequate irradiation
- Uncontrolled residual of extremely radiosensitive cancer
- Tumor condensed by irradiation
- Infiltrating and/or leukemic
- Deep and/or extensive cartilage invasion
- Paget disease of the breast
- Cancer developing in therapeutic burn scar and other scars
- Carcinoma degeneration in radiation dermatitis
- Cancer associated with acrodermatitis pyrodermatiformis
- Cancer developing in draining sinuses or fistulas
- Cancer developing in varicose ulcers
- Cancer developing in chronic acrodermatitis atrophicans
- Bleeding pain due to extension of cancer:
 1. Alcohol injection of ganglionic ganglion or its branches
 2. Surgical section of the branches or root of the trigeminal nerve
 3. Section of peripheral nerve trunks
 4. Subarachnoid alcohol injection
 5. Chordotomy
- Flap repair following excision:
 1. Flap graft
 2. Split graft
 3. Full thickness graft
 4. Skin graft
 5. Tube graft

Indications for Amputation

- Uncontrollable hemorrhage
- Widespread invasion of bone
- Invasion of joint
- Deep invasion of tendons or vessels
- Extensive suppuration
- Inoperable metastasis of popliteal or epitrochlear nodes associated with inoperable or operable groin or axillary nodes

Elective (surgery not essential)

- Papillary biopsies of all lesions
- Excision of small lesions
- Excision of some moderate-sized lesions
- Conservative (essentiality of surgery in every case a moot question)
- Metastasis from epidermoid carcinoma of the face
- Recurrence following inadequate irradiation
- Recurrence following surgical excision
- Inadequate excision of a suspected carcinoma

where wound has been closed by primary intention

Extramammary Paget's disease, Bowen's disease

Cancers developing from tar paraffin, etc.

Cancers developing in lupus vulgaris

Lesions of the skin of the sternal region and midline of the back

Lesions of axillae, legs, hands, and feet

Lesions in the aged with markedly trophic skin.

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CHANCROID

SYNONYMS *Ulcersmall soft chancre*

Acutely contagious, chancroid appears usually as a venereal—but nonsyphilitic—ulcer caused by the streptobacillus of Ducrey (*Haemophilus ducreyi*)

Etiology The organism is a clear centered short but thick, gram negative



Fig. 144 Chancroid. Of peak, with autoinoculation chancroid on anterior surface right thigh (visible to photograph)

bacillus, occurring singly or in more or less long chains. It is usually transmitted through coitus, but is exceedingly infectious and easily transmitted to healthy skins by direct inoculation. The sites of predilection for chancroid are the genitalia and vicinity. Chancroidal ulcers have been observed on fingers, eyelids, tongue and lower lip. The incubation period is short generally from twenty four to forty-eight hours, rarely

four days, and exceptionally seven days. Chancroid is more common in the genitally unclean.

Symptoms The first subjective sign is pain. On examination there is found at the painful point an easily bleeding, round or oval fairly regular ulcer with congested, well defined, perpendicular soft, detached borders. The irregular floor of the ulcer is covered with considerable greenish yellow pus. The ulcer usually enlarges rapidly reaching two to three times its original size in from four to five days, but even from the outset



Fig. 145 Chancroidal Ulcers.

involves the derm. A follicular type of chancroid is seen occasionally on the vulva and hairy areas of the genitalia, as well as a round, not very deep, so-called "dwarf" type. The follicular type

resembles simple staphylococcal folliculitis. Owing to their infectiousness, chancroids are apt to be multiple. Although chancroidal borders are always soft it is well to remember that the primary lesion of syphilis is not always indurated.



Fig. 146: Chancroid, Of lip, usually 1 cm

Chancroids tend to undergo spontaneous involution and heal with cicatrization as the organisms lose their virulence. The commonest complication is lymphangitis, especially of the dorsal surface of the penis and lymphadenopathy. Occasional complications are gangrene and phagedena.

Preputial chancroids often cause phimosis and paraphimosis.

Chancroidal Bubo This develops in about 30 per cent of the patients, and usually involves the inguinal nodes. Pain and tenderness and often fever accompany the adenopathy. The nodes enlarge, soften, and become adherent to the skin which reddens, breaks down and discharges a brownish pus from one

or several sinuses, the borders of which often assume a chancroidal character. The affection is thus often prolonged for from three to six months.

Triple Symptom Complex of Behcet In 1940 Behcet described a complex of symptoms characterized by oral aphthous ulcers, genital chancroidlike ulcers, and ocular changes (retinitis, iritis, iridocyclitis, choroiditis, and optic nerve change with partial or complete atrophy). The etiology is unknown. There is no specific therapy. Eruptions similar



Fig. 147 Chancroids. Multiple follicular chancroids in a patient with active gonorrhea. These chancroids are round, not very deep. They tend to spontaneous cure in several weeks.

to erythema nodosum have been described in some cases.

Diagnosis The clinical diagnosis of chancroid is usually easy. The laboratory aids are (1) smears and cultures for the *Ducrey bacillus*, which are often

negative (2) the automoculation test (3) the Ito-Reensterna intracutaneous test and (4) the complement fixation test.

In the *automoculation test*, the skin over the deltoid is punctured with a pointed instrument carrying some of the pus suspected of containing the organism. The pus should be obtained from beneath the chancroidal border. After the inoculation, the part is covered with a glass shield and examined forty-eight hours later. If positive there will be a small pinhead pustule or if not seen for four or five days, a crust, beneath which is a typical chancroidal pinhead or slightly larger ulcer. This test is liable to be negative when the pus is obtained from old chancroids or from those in which the streptobacilli have a low virulence.

The *Ito Reensterna intracutaneous test* employs a streptobacillary or Ducrey vaccine (Lederle) of which 0.1 cc. is injected intradermally. A red, edematous papule appears twenty-four hours later and reaches its maximum intensity in forty-eight hours in patients whose chancroids are eight or more days old. This test will be positive many years after the chancroid or bubo has healed. It may also be negative in the presence of chancroid.

Hyman and his co-workers believe that biopsy is the most efficient single diagnostic test, when used on primary genital lesions that are not too small.

Although the diagnosis of chancroid in itself is important, it is far more important to be certain that infection from *Treponema pallidum* is not also present. In this regard every chancroid is suspect until proved otherwise (by repeated observation of the lesion and

subsequent studies for blood serum changes for syphilis).

Treatment. The sulfonamides, especially sulfathiazole internally and locally in the form of a powder are specific. Sulfathiazole is given in 1-gm. (15.4-grain) doses four times daily for five days, and then reduced to a 0.5-gm.



Fig. 148 Multiple Chancroids.

(77-grain) dose four times daily for from seven to ten days. Ascending doses of *streptobacillary vaccine* are helpful. The *dimelcos vaccine* is generally used intramuscularly or intravenously beginning with 0.05 cc. ($\frac{1}{2}$ in.) and ascending to 0.2 cc. daily for from eight to ten days up to a total of 2 cc. *Circumcision* for lesions on the prepuce will prevent phimosis and paraphimosis. For early bubo intraglandular injections of a suspension of microcrystals of sulfathiazole are advised. Penicillin (20,000 units intramuscularly every three hours for six doses) should be tried in patients intolerant to the sulfonamides (O'Leary).

CHEILITIS

SYNONYMS: *Cheilitis glandularis apostomatosa myxodentitis labialis.*

Cheilitis is an inflammation of the lip

Varieties (1) Cheilitis exfoliativa
(2) cheilitis glandularis

Cheilitis Exfoliativa

Incidence This is commonly seen in skiers, mountain climbers and those ex-

posed to ultraviolet radiation. The term "cheilosis" has been used for the cheilitis seen in arboflavinosis

Pathology It is that of dermatitis.

Symptoms Erythema, edema, excoriation and crusting characterize the acute form of cheilitis. With the subsidence of the acute symptoms, edema diminishes and scaling appears. In the chronic form scaling and crusting are the only objective symptoms. As the scales and crusts fall off a glazed surface presents itself upon which new crusts form. Fissures may also be present. Subjective symptoms constitute pain, tenderness and burning sensations.



Fig. 149: Cheilitis Exfoliativa.

posed to ultraviolet radiation. The lower lip is more often involved than the upper lip

Etiology One of the common causes is exposure to ultraviolet radiation and sunlight (cheilitis actinica or sunburnt lip). Irritating lipstick, toothpastes, and mouth washes (cheilitis venenata) (perleche) may be causative factors. It may also be a symptom of hypersensitivity to orange juice, tomato juice, persimmons, menthol nail polish, and cigarettes. It is also associated with Plummer-Vinson syndrome, Pyorrhea alveolaris and seb-



Fig. 150 Cheilitis, Commisural or angular

Diagnosis Chronic cheilitis must be differentiated from precancerous keratosis. Precancerous keratosis is less inflammatory, drier and more localized than chronic cheilitis.

Prognosis It is resistant to treatment; however improvement may be obtained by proper treatment.

Prophylaxis Protection of lips from wind and sunlight by employing creams or lotions containing tannic acid, salol, quinine, or secalin is a prophylactic measure.

Treatment: Acute cheilitis is treated by the application of 0.25 per cent aqueous solution of silver nitrate or 1 per cent aqueous solution of aluminum acetate. Such ointments as 10 per cent application of aluminum acetate in aquaphor 2 per cent yellow oxide of mercury or whitmanol are equally beneficial.

Cheilitis Glandularis

Cheilitis glandularis is an inflammation of the lip where aberrant mucous glands are present on the vermillion border and on the inner surface of the lip.

Incidence It is seen in early or middle adult life.

Etiology: Cheilitis glandularis is a prenatally abnormality in which hypertrophy of aberrant mucous glands is the characteristic feature.

Pathology The pathology is characterized by enormous dilatation and hypertrophy of the mucous ducts and glands.

Symptoms The hypertrophic mucous glands can be definitely palpated beneath the mucous membrane. The orifices of the glands can be seen on the vermillion border of the lip the ducts are dilated. Droplets of mucus mark the orifice of the ducts. The lip is of normal size unless it becomes infected, in which event it becomes edematous and a purulent discharge is present. This condition is designated as cheilitis glandularis apostematosa. The subjective symptoms are tenderness and pain and a feeling of "heaviness" of the lip.

Complications This condition is definitely precancerous and a large percentage of them develop cancer of the lip.

Prognosis Cheilitis glandularis is not benefited by topical applications.

Treatment The most satisfactory treatment consists of removal of the individual glands, using a small cryosonic punch.

CHLOASMA

SYNONYMS *Liver spots, melasma, mack spots, Leberflecken, taches hepatiques.*

Chloasma is characterized by increased pigmentation of the skin occurring as variously sized and shaped, yellowish, brownish or black patches with ill defined margins.

Varieties Chloasma may be idiopathic or symptomatic.

Incidence It is rare in the male although more or less patchy discoloration occurs about the male crural and perineal region.

Etiology The cause may be either local or systemic. The idiopathic variety

results from various external agents, while the symptomatic variety is associated with some physiological abnormality or pathological change in various organs.

Pathology The pathology of chloasma shows an increase in the physiological pigment. In some instances, as in chronic skin diseases, the pigmentation is due to the coloring matter of the extravasated blood.

Symptoms. *Idiopathic Chloasma.* In this variety the pigmentary increase is

due to local or external agents. The various external agents include the following: pressure or friction from belts, trauma, scratching as in pediculosis corporis, application of heat (hot water bottles), sun's rays, actinotherapy and physiotherapy. The local application of counterirritants such as mustard, cantharides, and capsicum produce pigmentation of the skin. Pigmentation of the face and breast may be produced by the repeated application of cosmetics, toilet water (herpetic dermatitis) or face powder containing anilines. In these latter the sun's rays often precipitate the pigmentary reaction.

Specially interesting is the pigmentation resulting from prolonged hyperemia and irritation in consequence of the scratching induced by chronic irritation of the skin as seen in pruritus, dermatitis herpetiformis, and pediculosis. Cutaneous syphilides may also leave a pigmentary stain.

Symptomatic Chloasma. This is the most important variety and includes all forms of pigment deposits which occur as a consequence of various diseases. Examples of this are the pigmentation observed in association with tuberculosis (especially tuberculous of retroperitoneal glands), syphilis, cancer, sarcoma, malaria, Craves disease, and functional and organic affections of the utero-ovarian system.

In pregnancy, sterility, hysteria, chlorosis, and functional derangements of the uterus there often occurs a facial discoloration extending over the forehead and extending to the scalp in the form of a faint or decidedly yellowish or brownish tinge. In other cases, the discoloration is asymmetrical and macular involving the chin, lips, eyelids, and cheeks. Similar pigmentary changes oc-

cur on the linea alba, external genitalia, and nipples.

Diffuse or patchy macular pigmentation is occasionally seen in hypothyroid and hyperthyroid conditions. It is often present in cases of disturbance of the pituitary body (pituitary basophilism) and tumors of the adrenal cortex.

Generalized pigmentation is also associated with malaria, melanoepithelioma, cachexia, pellagra, and chronic alcoholism. It also occurs in the following dermatoses: lichen planus, psoriasis, eczema, pediculosis, leprosy, and scleroderma. A familial tendency for localized as well as generalized pigmentation has also been reported.

Diagnosis. Chloasma rarely causes any mistakes in diagnosis. It must be differentiated from tinea versicolor, icterus, carotinemia, and fixed drug eruptions. In tinea versicolor the causative organism (*Microsporon furfur*) can be readily demonstrated microscopically and there is usually a slight scabiness present. In icterus, a varying degree of pruritus is usually present and the color of icterus is a light yellow to a dark chrome. In carotinemia, the pigmentation predominates in the soles and palms and is more yellow than chloasma. Fixed drug eruptions are localized and a history of having taken such drugs as phenolphthalein is obtainable.

Prognosis. Pigmentation consequent upon local or external agents usually disappears after the causative agent is removed. Symptomatic chloasma is usually persistent, however the prognosis is dependent on whether the cause may be corrected.

Treatment. There is no specific remedy. Constitutional treatment is necessary in the symptomatic variety. Locally the application of solid carbon

dioxide for three seconds or the application of lemon juice is helpful. Other local remedies include the application of hydrogen peroxide a solution of corrosive sublimate (1:1000) lactic acid and peeling pastes.

George Andrews recommends the following cream:

Citric acid.	1.5
Formalin	1.5
Bis oxychloride	1.5
Ung. oil. rose q. s. ad.	30.0

CHONDRODERMATITIS NODULARIS CHRONICA HELICIS

SYNOYM *Painful nodule of external ear (Foster)*

Definition This term is used to designate a small painful tender nodule occurring on the rim of the ear.

Etiology The exact cause is not known. It is generally limited to the male and in those past middle life. The condition is not rare and appears to be a low grade chronic, inflammatory disease involving the corium and cartilage.

Symptoms Either one or both ears may be affected. The patient usually complains of being unable to sleep with the affected ear pressed against the pillow. On examination, a firm, ovoid, flat topped pinhead or larger-sized, well defined nodule is seen on the free border of the helix at the upper part of the auricle. They are very tender immovable skin-colored or pinkish embedded in, or elevated several millimeters above, the level of the skin. The center of the nodule often shows a keratotic center. Removal of this adherent scale exposes a depressive sometimes slight ulceration.

Diagnosis They must be differentiated from topoi epitheliomata and keratosis. Topoi are painless, unless ulcerated or secondarily infected and show sodium urate crystals on puncture. An epithelioma is a progressive lesion with firm, elevated rounded borders and waxy color. Sooner or later it shows central surface erosion and later ulceration. Keratosis are scalp flat, nonnodular lesions occurring on an inflammatory base which bleed on removal of the scales.

Prognosis The lesions reach a certain size and then remain stationary. Spontaneous regression occasionally occurs especially if pressure, as from a telephone ear piece, is avoided. Malignant degeneration has not been observed.



Fig. 151 Chondrodermatitis Nodularis Chronica Helicis. In woman and at an unusual site. (Courtesy of Dr. Kurt Jasslik.)

Treatment Excision or electrocoagulation of the lesion is curative. Curettage under procaine anesthesia, followed by cauterization, occasionally is successful. The lesions may also be destroyed by solid carbon-dioxide pencil. Roentgen-ray therapy usually gives relief from pain, although it is apt to be temporary.

COCCIDIOIDAL GRANULOMA

SYNONYMS: *Desert fever valley fever California disease.*

Coccidioidal granuloma is an acute coccidioidal disease which is endemic in Southern California parts of Texas, Arizona and New Mexico. It occurs sporadically in other parts of the world.

Varieties. There are two varieties, acute and chronic.

Incidence. It occurs both in human beings and animals.

Etiology. Coccidioidomycosis granuloma is caused by infection with *Coccidioides immitis*, a yeast like organism. Infection takes place through abrasions in the skin or through inhalations of spores. The disease is often transmitted by the handling of raw fruit. It occurs frequently among grape pickers. It is believed that insects feeding on contaminated soil may transmit the disease to men and animals. When the disease is caused by inhalation of spores, the primary lesion develops in the lung.

Pathology. The structure of coccidioidal granuloma is similar to that of infectious granuloma. The causative organism resembles *Blastomyces* except that *Coccidioides immitis* is larger, does not show budding forms and multiplies by endogenous spore formation. Recently, however, De Lamiater has demonstrated budding (yeast like) forms in the tissues. It grows readily on nearly all culture media. The organism may be demonstrated in smears from the pus of cutaneous lesions, from the sputum, spinal fluid, etc.

Symptoms. It is essentially a systemic disease; cutaneous symptoms occurring only in the minority of cases. All organs and body tissues except the gastrointestinal tract may become involved. The early cutaneous lesions are usually located on the hands and arms, less frequently on other exposed por-

tions. The primary lesions are nodules which eventually suppurate and form numerous sinuses resembling scrofuloderma. Lesions simulating mycosis fungoides or gumma may also be present. The local lesions of coccidioidal granu-



Fig. 152: Coccidioidal Granuloma. Showing solitary ulcerated nodule. (Courtesy of Dr. H. E. Miller, San Francisco.)

loma may remain for years before dissemination via the lymph or blood stream occurs. If the cutaneous lesions are secondary to systemic involvement, variously sized subcutaneous soft nodules or deep abscesses may occur. If the lungs are involved the symptoms are those of acute bronchitis or bronchopneumonia. The subjective symptoms do not differ from those of systemic blastomycosis.

In the acute variety death occurs in a few months, while in the chronic form the disease may last for years.

Diagnosis - *Coccidioidomycosis* must be differentiated from *blastomycosis*, *tuberculosis*, and *syphilis*. The demonstration of the causative organism, microscopically culturally or by animal inoculation (preferably mice) settles all arguments as to diagnosis. In mice there develop numerous, easily identifiable, endospore-lating spherules (sporangia) as well as budding cells. *Blastomyces* produce only budding cells.

Suspected cases of *blastomycosis* that fail to improve under iodides may be possible cases of *coccidioidal granuloma*.

Prognosis The mortality rate of the disease in California according to Ernest C. Dickson, is slightly less than 80 per

cent. This disease is, however usually fatal.

Treatment - *Coccidioidin* (a vaccine prepared from many strains of this organism or autogenously) given intravenously is occasionally helpful. The initial dose, according to Jacobsen should be 0.05 cc., and each succeeding injection should be increased 0.05 cc. Twelve to fifteen injections are given at eight to fourteen day intervals, with the dose held stationary when reactions occur. Two or more courses may be given with a six to eight-week rest period between courses. *Antimony* and *potassium tartrate roentgen therapy gentian violet*, and *thymol* are all worthy of trial.

COLLOID MILIUM

SYNONYMS *Hyaloma*, *colloid degeneration*, *hyaloma der Haut*.

Colloid milium is a cutaneous affection characterized by pinhead to split pea-sized translucent nodules, of a yellowish color and usually occurring on the upper part of the face.

Incidence It occurs in both sexes, although it is usually met in men over forty; however several cases have been reported occurring in children aged nine and twelve.



Fig. 153 Colloid Milium. (Courtesy of Dr. Stuart C. Way)

Etiology The cause is unknown, but exposure to heat sunlight and the elements are thought to be possible factors.

Pathology The principal histopathologic changes involve the connective tissue in the corium especially in the periglandular region. This consists of hyalin like or colloid degeneration of col-



Fig. 151: Colloid Milium. Note lemon-yellow lesion on eyelid, translucent in appearance which is suggestive of molecular formation.

lagen and elastic fibers. Some clinicians consider it the result of sebaceous gland degeneration hence the name colloid milium is justly applied.

Symptoms Colloid milium is characterized by rounded yellowish translucent nodules ranging in size from a pinhead to a pea. The sites of predilection are the forehead about the orbit nose and cheeks. An extensive case has been reported by Jager in which nodules were situated over the nose cheeks ears neck and dorsum of the hands. They develop slowly often in groups though the individual papules remain distinct

even when two or more unite. The nodules are of firm consistency and of pale or orange-yellow color suggesting vesicles. Some of them may be surrounded by slight telangiectasis. Incision and expression of the contents reveals a small amount of gelatinous translucent yellow or brown substance. Subjective symptoms are absent.

Diagnosis The disease must be differentiated from xanthoma hydrocystoma multiple benign cystic epithelioma pseudoxanthoma elasticum senile elastosis and adenoma sebaceum. Xanthoma is nontranslucent in appearance soft to the touch and usually limited to the eyelids. The lesions of hydrocystoma lack the yellow color of colloid milium and on pricking a fluid exudes. Multiple benign cystic epithelioma appears in early life it is often familial. It lacks the yellow color of colloid milium. In pseudoxanthoma elasticum the flat yellowish nodules are on the neck trunk and flexor surfaces about the joints; histologically there is clumping and splitting of the elastic fibers. In senile elastosis there is no nodule formation. The skin is yellow and thickened. The diagnostic features of adenoma sebaceum are the early appearance the region involved and the associated telangiectasis. Colloid milium lesions are free from surface-dilated capillaries and are more distinctly yellow in color. Histological examination may be necessary in some cases.

Treatment Treatment consists of the destruction of the nodules either by curettage or electrolysis. In a patient seen by Stuart Way there was marked improvement following prolonged use of large doses of vitamin C fruits and green vegetables.



Fig. 185 Acute Dermatitis Venenosa. (Courtesy of Dr. Carroll S. Wright.)

Contact Dermatitis

CONTACT DERMATITIS

SYNONYMS *Dermatitis venenata, eczematous dermatitis, occupational dermatitis, industrial dermatitis.*

Contact dermatitis is an inflammation of the skin caused by cutaneous irritants, either animal, vegetable mineral, or chemical.

Varieties Contact dermatitis may be either acute or chronic. An acute contact dermatitis is characterized by erythema, edema, vesicles, and papules. A chronic dermatitis is characterized by infiltration and lichenification.

Incidence Individuals with thin blond skin are more liable to contact dermatitis than those with thick oily skin. Negroes are, therefore, less susceptible to the action of skin irritants than the white man.

Various portions of the skin of the same individual vary in their susceptibility to irritation. The inner aspect of the forearms and the anterior portion of the body are much more liable to contact dermatitis than are other parts of the body. Young people, especially females, suffer from contact dermatitis more often than older ones. There is also more industrial dermatitis in warm weather than in cold because less clothing is worn and close contact with external irritants is therefore possible. Persons who perspire freely more often develop contact dermatitis. Diet influences the hydrogen ion concentration of the perspiration and may therefore be a factor.

Etiology Contact dermatitis may be caused by any number of cutaneous irritants. The eruption always begins at the place of contact with the offending agent and generally within one to five days after the contact. The concentration of the irritants, the continuous or intermittent contact and the condition under

which the exposure occurs differentiate two types of contact dermatitis namely a nonsensitization and a sensitization dermatitis (J. G. Downing).

Nonsensitization dermatitis is caused by a cutaneous irritant that in given concentrations and surroundings affects nearly all types of skin. An example of such a dermatitis is the reaction of the skin to powerful chemicals such as phenol, chromic acid, lye, nitric acid etc. These are called primary irritants.

Sensitization contact dermatitis is the name applied to a dermatitis resulting from repeated exposures to substances normally harmless. These have been termed specific irritants and occur in many trades. The initial exposure may not produce a reaction but subsequent exposures cause a dermatitis at the site of contact or even a generalized reaction. Among the common causes of sensitization contact dermatitis are plants, hair dyes, other hair applications, clothing dyes, depilatories, nail polish, lipstick, cosmetics, dress shields, adhesive plaster novocaine and soap. The list includes many hundreds of substances.

Gay has grouped the irritants as follows:

(A) CHEMICAL PRIMARY IRRITANTS
GENERAL IRRITANTS

Inorganic—

Acids and Salts	{	Bulphuric
		Nitric
		Arsonic
Salts of Irritant Metals	{	Mercuric
		Chromic
		Arsonic
Alkalis	{	Sodium Hydroxide and Carbonate
		Potassium Hydroxide and Carbonate
		(Sensitization usually an important factor)

Organic.

Acids, Anhydrides, Salt of

Acetic
Carbolic
Formic
Oxalic
Picric
Salicylic

Solvent

Hydrogenated Phenol
Turpentine
Formaldehyde
Xylol
Chloroform

(B) SPECIFIC IRRITANTS WHICH CAUSE DERMATITIS IN THOSE SENSITIZED BY PREVIOUS EXPOSURE

Explosives

Gases

Cosmetics

Rubber Compounds

Oils (Vegetable and Mineral)

Insecticides and Fungicides
Fabric Dyes

Fu Dyes

Resins and Waxes

Leather Dyes

Bacterial Agents
Parasites
Fungi

Photosensitizers

Phot. Developers

Certain individuals manifest a distinct susceptibility to cutaneous irritants, while in others this predisposition is due to scratching hyperidrosis burns, or traumatism. Sensitivity to a single cutaneous irritant may be followed by a polysensitivity in which case the individual is sensitive to many substances. Persons may be exposed to poison ivy and other allergens for years without manifesting any cutaneous reaction when after subsequent contact a dermatitis may suddenly develop. Why this sensitivity suddenly appears is unknown the answer is a conjecture perhaps coincident fungus or bacterial infection is the trigger which brings about this sensitivity. In fact anything which lowers the cutaneous threshold of resistance may be the precipitating factor in the production of a contact dermatitis.

On the other hand a cutaneous, sensitive state of long duration may disappear spontaneously. In others, as in Japanese lacquer workers, a state of cutaneous immunity may follow prolonged exposure and repeated attacks of dermatitis.

Pathology Edema vesiculation and necrosis of the epidermis are the basic pathologic changes in contact dermatitis. Mesodermal inflammation with capillary dilation edema and leukocytic infiltration of the dermis also occurs. The pathology varies with the degree of reactivity chronicity frequency of contact and the secondary effects of infection.

Plant and Weed Dermatitis

Contact with plants and weeds is among the commonest cause of dermatitis. The usual characteristics of plant and weed dermatitis are the appearance of linear and grouped vesicles accompanied by marked edema.

Varieties Nearly all plants and trees have been reported as causing a dermatitis in susceptible persons. The objective symptoms resemble each other to a great extent however they may differ in their appearance some are less vesicular and more erythematous than others. The plants which are the most likely to produce dermatitis are poison ivy (*Rhus toxicodendron*) primrose (*Prunella obconica*) poison oak (*Rhus diversiloba*) poison sumac (*Rhus vernix*) ragweed (*Ambrosia trifida*) chrysanthemum and pyrethrum (this is prepared from chrysanthemums and is contained in many roach powders and fly sprays). It appears probable from chemical and biological evidence that the same chemical substance is the active agent in the saps of the poisonous varieties of ivy oak sumac and the Japanese lac trees. It is a nonvolatile oil a benzene compound and has been called "urushiol" (toxicodendrol" by some authors). Certain oxidases, especially mushroom tyrosinase (Sizer and Prokesch) can render it innocuous, but the clinical value of these remains to be determined.

The onset of plant and weed derma

titis occurs a few hours or days after exposure. The areas of involvement are the face, neck, wrists, and hands. The dermatitis spreads by autoinoculation, especially in scratch marks, and in this manner linear lesions are produced

Rhus Dermatitis

SYNONYM *Ivy poisoning.*

Etiology *Rhus dermatitis* is caused by contact with the poisonous sap pres-

from burning plants to cause dermatitis in sensitive persons

In some cases, a latent period intervenes between the exposure and the appearance of hypersensitivity. This latent period may be a few days or many years. Previous contact with plants and weeds is, however, necessary for the development of sensitization. The sensitivity may or may not be permanent. As with all contact dermatitis, no serum

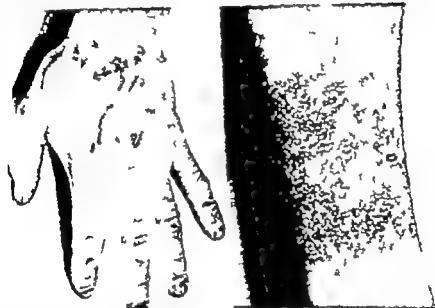


Fig. 156 Left: *Dermatitis Venenata* (poison ivy). Note the characteristic streaks of eczema-like. Right: *Dermatitis Venenata* (poison oak). (Courtesy of Dr. Jacques P. Guéguen.)

ent in the leaves and other visible parts of the poison ivy plant. The sticky sap carried on other individuals' clothing, on a unal, tools, or present on other objects, accounts for the development of *rhus dermatitis* on those who have no direct contact with the plant. The common conception that the oleo resin of poison ivy as well as other plants and weed is oleo resin is erroneous; however, enough of the oil can be carried on dust and soot particles and in smok-

antidies have been satisfactorily demonstrated and the fluid from the vesicles and blisters is harmless for normal skin (Corson).

Symptoms: *Rhus dermatitis* is characterized by vesicles which appear in groups or in a linear arrangement. The hands, feet, face, and genital regions are the parts usually affected. The dermatitis is transmitted by the fingers to the various parts of the body. It usually runs an acute course and subsides spon-

taneously in the course of a few weeks to a month. Recurrences are frequent while repeated attacks do not confer an immunity.

Subjective symptoms consist of marked itching and burning sensations. Poison ivy rarely affects the mucous membranes. Seymour H. Silvers reports a



Fig. 157: Chemical Contact Dermatitis.
Due to poison ivy

case of a woman who chewed poison ivy leaves in the hope of desensitizing herself. Two days after chewing several leaves of the poison ivy her mouth became sore and a vesicular eruption developed around the mouth and on the hands. During the next few days the mouth soreness increased and extended deep into the pharynx. Pain followed movements of tongue and cheek. The mucous membrane of the tongue, cheek, palate and pharynx was tender, grayish in color and edematous. The anal region also became red and extremely sore. After three days treatment oral symptoms disappeared and eating became painless. The itching pain and redness of the anal region persisted the longest.

Ragweed

Dermatitis produced by the several varieties of ragweed is characterized by

redness, edema, itching of the eyelids, and erythema of the exposed surfaces, such as hands, wrists, and ankles. It usually manifests itself through the months of July, August and September and disappears with the first frost. Children and the aged are rarely affected. It is commonly observed in farmers. It may be acute but often consists of chronic, crusted, lichenoid lesions, sharply limited to the face, neck and extremities. Patch tests with ragweed extract are important diagnostically. Change of occupation is often necessary.

Poison Sumac, Dogwood, and Ash

These plants are occasionally productive of a dermatitis. It resembles rhus dermatitis but has a tendency to be more erythematous and less vesicular. It also occurs later in the summer.

Primrose Dermatitis

The active chemical substance causing dermatitis with *Primula obconica* is called "primulin."

Symptoms of primrose dermatitis usually develop from eight to ten hours after handling the plant. It usually begins as a punctate or diffuse erythema on the index finger and thumb of the right hand or on the eyelids and anterior portions of the neck. From the above mentioned areas it may spread to other parts of the body. The original objective symptoms are usually followed by edema, vesiculation and desquamation.

Certain woods, such as lacquer tree, mahogany and Danzig oak may also cause dermatitis in susceptible persons. The toxicity of the saps of these woods and of poison ivy, swamp sumac and lacquered products is lessened by aging, oxidation and exposure to air. However, thick deposits of dried sap may

resist oxidation and loss of toxicity for many moths.

Dhobie (Laundryman's) Mark Dermatitis

This dermatitis is seen in India and is due to contact with laundry marks. The indelible ink used comes from the poisonous black oil of the nut from a tree (*Semecarpus anacardium*)

Cosmetic Dermatitis

Dermatitis of the face often follows the application of face powders or lipstick. At the site of application (face, neck, and lips) a feeling of dryness and tension first occurs and this is followed by erythema. Louis E. Pierini states that a chronic pigmented dermatosis may follow this reaction and considers this also due to sensitization. It may develop a few hours after the application or not for months. Sensitization to rouge developed in a woman who had used that brand of rouge for thirteen years. Primary irritation from cosmetics is rarely observed today since chemicals now used in cosmetics are harmless to nearly all people. Most cases of dermatitis result from cutaneous hypersensitivity.

The coloring matter in rouge, lipstick, and face powders is eosin, carmine, or other aniline dyes. The aniline dyes, especially the group of orange dyes, enjoy the reputation of being cancerogenic and their use should, therefore, be interdicted.

Match Box Dermatitis

This is characterized by the occurrence of a circumscribed inflammatory patch beneath the pocket where the matches are carried. The involved area is red, rough, infiltrated, and scaly. Occasionally edema of the scrotum may be a symptom. These patients are hyper-

sensitive to the phosphorus on the box or package of safety matches.

Emotional Dermatitis

This is characterized by intermittent eruptions of small vesicles on the palmar surface of several fingers. Fissures occasionally occur on these same areas. It is an asymmetrical eruption and appears and disappears at varying intervals. These lesions are due to emotional factors either of an exhaustion-fatigue type, or just plain emotional instability due to unsatisfactory business or home relations. It has a definite psychogenic background. A psychiatrist should be consulted in these cases (see also Cutaneous Neuroses, p 248).

Nail-Polish Dermatitis

The characteristic picture of nail-polish dermatitis is patches of dry scaling dermatitis on the face, sides of the neck, chin, eyelids, and occasionally on the trunk. The fingers and the hands are not involved. The patches are irregular in shape, and vary in size from a quarter to a half dollar. Pruritus is sometimes a prominent symptom.

The allergen may be the dye, the solvent, or the perfume in the nail polish.

Industrial Dermatitis

SYNONYMS Trade eczema, occupational dermatitis.

The National Committee of Industrial Dermatoses has agreed on the following definition of industrial dermatitis. An occupational dermatosis is a pathological condition of the skin for which occupational exposure can be shown to be a major causal or contributory factor."

Dr C. Guy Lane suggests that the following information should be elicited in arriving at the diagnosis of industrial dermatitis:

1 That the time relation between the exposure to the agent and the onset of the dermatosis is correct for that particular disease

2 That the individual has an occupation with a high cutaneous morbidity

3 That he has been working in contact with an agent known to have produced similar changes in the skin

4 That the site of onset of the skin disease coincides with the site of maximum exposure or trauma

5 That some of his fellow workers with the same agent have similar manifestations

6 That no possible exposure outside his occupation has been found to be an agent which could cause similar lesions

7 That if the diagnosis is dermatitis the history of multiple attacks, coming after exposure and reexposure to an agent followed by improvement and clearing after cessation of exposure constitutes most convincing evidence that the occupational factor is a cause

Incidence Dr Louis Schwartz, of the United States Public Health Service believes that 1 per cent of the industrial workers in the United States are actually affected with dermatitis, and that the continual introduction of new chemicals and new processes may be expected to cause an increase in this percentage

Most of the cases occur among metal workers, machinists, domestic workers, and handlers of food

Etiology The United States Public Health Service tabulates the following causes

- 1 Petroleum oil and greases
- 2 Alkalies, including cement and concrete
- 3 Solvents
- 4 Chromic acid and salts.
- 5 Metals and metal plating
- 6 Dyes.

7 Plants

8 Rubber and its compounds.

9 Paints and varnishes.

10 Synthetic resin

Symptoms The immediate effect of contact dermatitis is characterized by erythema, vesiculation, bullous dermatitis, or a destructive burn

The clinical appearance of the eruption is subject to great variation and depends upon the portion of the skin that is sensitized to the allergen as well as the nature of the allergen. However if recovery or marked improvement does not follow six to eight weeks absence from work one is justified in excluding a contact in that person's occupation as a cause for the existing dermatitis. The wearing of impervious sleeves and gloves may be both diagnostic and therapeutic

Dermatitis Due to Acids, Alkalies, and Some Metallic Salts

Acids Hydrochloric acid burns are encountered in those who handle or transport the acid, and in plumbers and those who work in galvanizing or implate factories. Hydrochloric acid produces burns which are less deep and more apt to form blisters than burns from sulfuric and nitric acids. Nitric acid is a powerful oxidizing substance and produces deep burns. The affected parts are yellow in color. Such injuries occur in those who manufacture the acid or use it in the making of explosives in laboratories. Sulfuric acid produces a brownish charring of the skin and ulcerations that heal slowly. Sulfuric acid is used more widely than any other acid in industry being handled principally by brass or iron workers or by those who work with copper or bronze. Other strong acids capable of producing burn include acetic, salicylic, hydrofluoric and oxalic acids, and also phenol

Alkalis Alkalis penetrate the skin and effect a solution of the tissues. Strong solutions are corrosive and the effects should be neutralized immediately by the application of weak acids. Alkali itch, a common form of occupational dermatitis, is produced by patent cleansers. It also occurs outside of in-

and unslaked lime. Water glass (sodium silicate) has caustic properties and is used chiefly in the manufacture of soap, in paper sizing, and as a preservative for eggs.

Metallic Salts Metallic salts have an irritating and corrosive effect upon the skin. White considers the chlorides



Fig. 128 Dermatitis. Of alkalis.

dusts in those who have dry skins and bathe too much in the latter case, the condition is called bath pruritus.

Alkalis are also used in manufacturing soap, affecting those who handle greases and textiles. The principal compounds are sodium and potassium hydroxide the alkaline sulfides (depilatories) the hypochlorites, and slaked

and nitrites the most corrosive salts, and next the sulfites, sulfides, and sulfates. Mercuric chloride in weak solution (1:5000) may be irritating, causing eruptions chiefly among surgeons, nurses, and taxidermists. Fulminate of mercury produces erythematous and papular eruptions conjunctivitis, and swelling of the eyelids. Nitrate of mercury produces

dermatitis among felt hat workers, and those who do etching, embossing or art metal work. Persons manufacturing thermometers, those handling furs, using amalgams, making barometers, fire-gilding or using solder for dry batteries are exposed to mercury and may develop dermatitis.

Phosphorus Phosphorus may produce a dermatitis or a burn. Match box dermatitis is a well known example. Phosphorus is used chiefly in the manufacture of fireworks and rat poisons.

Silver Silver may cause a localized or diffuse bluish black discoloration which is known as argyria. It follows long continued medical use of silver nitrate or other silver compounds. Industrial cases have been reported among silver smiths and those who work with silver leaf and pearl beads.

Antimony Salts of antimony may produce a papular pustular or follicular dermatitis. It is rarely encountered in industry.

Arsenic Arsenic is one of the most common chemical causes of dermatitis, being observed in those who mine, extract or refine copper, arsenic pyrites or other arsenical ores, and in those who have contact with the artificial coloring of wallpaper, flowers, and chalk. Arsenical compounds are also used in dyeing fabrics and domestic articles for the preservation of animal skins and hides, and embalming. Arsenic is an ingredient of some disinfectants and weed exterminators, and is used in sheep-dip factories, and in the making of fly paper. It is encountered in submarine work, establishments where radio and automobile batteries are charged in electroplating works, chemical factories for the manufacture of sulfuric and other acids, insecticide factories, and general chemical factories, printing establishments

where gilt or bronze powder is used, in farming and in gardening. Woolen goods which are sprayed with a solution of an arsenical compound are also a source. Among those who are liable to arsenical dermatitis in the course of their occupation are glucose and candy factory workers, those who use sugar and dextrin, bookbinders, fruit handlers, farmers who handle raw furs, machinists, and metal workers who handle brass, copper and zinc. Excessive quantities of arsenic have been found frequently in gelatin, raw and canned fruits and vegetables, adulterated food coloring and confectionery, sea food, in baking powders, egg powders and self raising flours due to impure acid phosphate and in cocoas and other chocolate products in the manufacture of which potash is used. The present method of disposing of the boll weevil through the use of calcium arsenate accounts for the presence of arsenic in cotton.

Chromium Chromic acid and the bichromates are strongly corrosive and irritative to the skin. Employees in chromatic works, tanners, French polishers, photostat workers, and those concerned with the bleaching of crude oils, tallow, and fats are subject to chrome dermatitis. The skin changes are multiform, ranging from a mild follicular dermatitis to widespread nodular and crusted eruptions. The sites of predilection are exposed parts of the body. The chrome hole or sore is a characteristic lesion and occurs upon the backs of the hands and forearms, usually beginning about a hair follicle or in the creases of the knuckles, or in the finger webs. The hole begins in a small abrasion which deepens and widens, eventually forming a conical indolent ulceration. Perforation of the nasal septum occasionally occurs in chromium workers.

Dermatitis Due to Various Dusts

Many mineral, chemical, and vegetable dusts produce dermatoses and inflammations of the mucosa. These dusts are usually partially soluble in the skin secretions. Some of them undergo hydrolysis and then become irritating. The amount of moisture in the atmosphere and the heat of the workrooms are conditions which make even a normal person's skin damp and sticky and powders are the more likely to adhere. A combination of mechanical and chemical irritation usually exists in dermatitis due to this cause. The dust sometimes clogs the pores, obstructing the outflow of sebum and sweat. Dust may also contain spores which provoke a dermatitis by growing upon and invading the skin or by acting as an allergen. The skin manifestations tend to be present on exposed parts, especially the hands, face, and neck; however saturation of the clothing with dust may involve such apposed surfaces as the armpits and groins.

Lime dust causes eruptions in plasterers or those who spread fertilizers. It is also observed in the smelting, bleaching and dyeing trades. Zinc dust produces acneiform eruptions. Dust of nickel and brass and ores containing arsenic may produce a follicular dermatitis.

Silicic dust is present in scouring soap. Workers in silicate cotton are subject to dermatitis from irritation by the spicules. Acetylene gas from calcium carbide in contact with moist skin forms calcium hydrate, which produces a dermatitis and necrosis leading to punched-out ulcers. Calcium cyanide is a light powder which provokes dermatitis or ulceration, and a peculiar systemic intoxication. Dusts

of high explosives occasionally produce a dermatitis which at first is erythematous, vesicular and localized, but later becomes widespread and associated with the exfoliation of the epidermis. Hamilton believes that skin affections are most prevalent in dinitrochlorobenzene workers; next in decreasing ratio in tetryl, T.N.T. and picric acid workers.

Picric acid is more active when in solution or in an ointment than in the form of a dust. Although picric acid dressings are generally harmless, occasional instances of extreme hypersusceptibility are encountered. A variety of war gases may cause eruptions.

The dusts of a large number of woods incite itching and dermatitis among persons working with them. Also some dry vegetable powders composed of minute grains and fibers, or containing spores and the mycelia of parasitic fungi, may have similar effects. Dust from cinchona bark, quinine, aruca, teak wood, pyrethrum, or Black Flag (an insecticide containing pyrethrum) often produces widespread dermatitis. Many eruptions due to insect powders belong in this category. Tobacco dust in cigar factories may also become a responsible factor. Powdered ornitho root and lycopodium (ingredients in face powder) may cause eruptions in sensitive persons.

Dermatitis Due to Various Hydrocarbons

Many hydrocarbons produce skin eruptions. Raw petroleum causes generalized itching or folliculitis and acneiform eruptions. The latter are frequently pustular due to a predisposition of the inflamed follicles to invasion by cocci. Lubricating oils and cutting emulsions are causes of similar cutaneous lesions. When they are used over and

over without filtering or sterilizing the fine metal fragments cause minute abrasions in the skin and infections by pyogenic micro-organisms are passed on from one worker to another through the medium of contaminated oil. Cutting oils are used both to cool and lubricate cutting tools. Two types are used soluble and insoluble. The soluble oils contain a sulfonated mineral oil soap resin sulfonic acids and a preservative such as cresol phenol or nitrobenzene. The insoluble cutting oils contain chiefly mineral oil some arsenical and vegetable oils, sulfur chlorine and in some a preservative like phenol. The refined fractions from petroleum are less irritating than the unrefined products, although benzene naphtha and carbon disulfide may cause a mild dermatitis. Briquette makers develop dermatitis as a result of a tarry residue from petroleum used in their trade. Workers in paraffin frequently develop a dermatitis which may cause pustules, keratoses, and ulcerations. In shale oil workers there develops an erythematous follicular eruption that eventually leads to keratoses and occasionally results in carcinoma. White estimates that 50 per cent of shale oil workers suffer from skin troubles.

Impure and low grade paraffins and mineral oils cause similar skin eruptions. The skin changes are characterized by black-capped projecting follicles that look like comedones, itching papules, and pustules. Gradually keratoses appear and after many years some of these are the sites of carcinoma. The sites of predilection are the forearms. Petrolatum dermatitis is characterized by a verrucous thickening of the skin. It is caused by prolonged contact with impure petrolatum and occasionally lubricating oil. A second or follicular type also occurs on the anterior and inner aspect

of the thighs in which erythematous horny nodules are present. There are no comedones present and the lesions are separated by apparently normal skin. A third type called acne cornes consists of follicular keratosis and pigmentation and was described by Oppenheim as an occupational dermatitis due to tar unrefined tar oils, and paraffin. It involves chiefly the dorsal aspects of the fingers and hands, the arms, the legs, the face, and the thorax. The subjects are usually railroad workers, engineers, mechanics, and stokers. The lesions are follicular horny papules often black, and are associated at first with a follicular erythema and later with a dirty brownish or purplish spotty pigmentation, which in severe cases becomes widespread being especially marked about the genitals. This syndrome may simulate pityriasis rubra pilaris or lichen spinulosus.

Mule spinners cancer has been an important problem in the cotton industry in England only a few cases have been reported in this country. Low grade oils used for the lubrication of the machine produce inflammatory and warty changes in the skin of the scrotum, which part is chiefly subjected to irritation. Neglect of personal cleanliness strongly predisposes to this complaint.

Leitch and Kennaway and others have experimentally produced paraffin and tar cancer in mice. In man it occurs principally in employees of gas works. Coal tar and pitch and many of their derivatives produce occupational dermatoses, specifically stimulating epithelial proliferation. Naphthalene, creosote, anthracene, and related products are responsible for skin eruptions. Anthracene oils and acridin are used as rubber substitutes in some telephone and radio receivers and have been the cause of dermatitis of the ears. In the manufacture of

chlorase the workers develop an acneiform dermatitis and a folliculitis (chlor acne) with small black plugs and papules. The site of predilection is the face.

Dermatitis Due to Dye, Pyroxilin Rubber and Other Substances

Dermatitis occurs in those engaged in the manufacture and use of synthetic dyes, chiefly paraphenylenediamine (Ural) which is used for hair and fur dyes.

Distribution is an important diagnostic feature of dye dermatitis. The eruptions due to hair dyes usually begin with itching, redness, and puffiness of the upper eyelids, tops of the ears, sides of the forehead, and back of neck, being most pronounced about the follicles. The affected areas become lichenified or scaly and the eruption spreads to other parts, especially the bends of the elbows. Eye-lash dyes cause persistent pruritic and swollen eyelids, and may cause serious eye injuries. The dye and the pyroxilin in artificial tortoise shell spectacles may cause dermatitis. The distribution of dye dermatitis due to fur collars and cuffs occurs first under the chin, on the front of the neck, over the presternal area, and on the wrists.

The practice of coloring citrus fruit by means of dyestuffs, aniline and other substances, and the use of various chemicals to ripen artificially and to preserve the fruit for distant markets have also produced dermatoses. It is estimated that a large proportion of the Florida oranges in the New York market are artificially colored. Metal which is used in photography may also produce dermatitis on being the face and hands.

Leather dye dermatitis usually occurs on the feet and is apt to be aggravated in warm weather. However other agents used in the manufacture of shoes (such

as the glue felt canvas, and antimildew preparation) may be causative factors.

Chemical permanent waving of the hair is at times the cause of a severe widespread dermatosis. Henna is incorporated in some hair dyes and nail paints, and causes eruptions that clinically resemble those described due to urushiol. Eyebrow pencils that contain mastic or artificial coloring from coal tar products may produce a redness and swelling of the upper lids. Inks and polishes are sources of dermatitis. Depilatories and shaving creams containing barium sulfide or excessive amounts of alkali are common causes of skin eruptions. Among the insecticides roach powders and mosquito sprays frequently provoke a dermatitis. Idiosyncrasy to perfume is occasionally encountered. Dermatitis due to rubber articles may be caused by the accelerator used in curing the rubber such as hexamethyl enetetramine, guanidine, tetramethylthiourea disulfide and others, or to wild rubber. Adhesive plaster dermatitis results from these ingredients.

Soot lamp black and peat produce dermatitis of a dry scaly character which in the course of time forms warty outgrowths and which cause cancer. Chinnee-sweeps cancer was first described by Pott in 1775. The cancer occurs upon a soot wart and is usually located upon the scrotum where the soot, sebum, and dirt collect in the folds of the skin. This form of cancer is much less prevalent than formerly due partly to greater cleanliness in trade methods. It is unknown outside of England.

Treatment

The causal treatment of contact dermatitis consists in eliminating or reducing the susceptibility to eczematous or allergic disease. An attempt to desensit-

sensitivity of the skin area tested to the tested material. It does not necessarily mean that the dermatitis is due to that substance. A negative means only that the area tested is not sensitive to the tested material (other areas, and especially the affected area, may be). That is



Fig. 160 Chemical Contact Dermatitis. Due to accidental application of a lilac lotion

why such tests should be made as close to the affected area as possible)

For these reasons it seems inadvisable to advocate the routine or extensive use of patch tests by the nonspecialist. The use of certain forms of patch testing is relatively simple and safe and may be of assistance to general physicians who are attempting to find causes of eczema under their treatment. For example a $\frac{1}{4}$ or $\frac{3}{8}$ inch square of the shoe leather or dress under suspicion or of any other article of wearing apparel can be moistened with water laid on an unaffected area of skin covered with a larger piece of impermeable material (oiled silk) held in place with a larger piece of adhe-

sive tape and left on for twenty-four to forty-eight hours. It is then removed, and the site is carefully inspected to see whether an eczematous reaction has been produced in the area on which the material rested. Similar tests can be performed with the artificial resins and plastics and with varnished painted, or metal objects by placing scrapings from their surfaces on a moistened square of linen and applying this square under oiled silk and adhesive tape in the manner already described. In the case of plants, fruits, etc. a small portion of the leaf petal flower or fruit may be applied under the oiled silk and adhesive tape. When cosmetics (cleaning lotion, powder lipstick) are suspected, these



Fig. 161 Dermatitis. Of nasal bridge and cheeks. Note involved area proximal to eyeglasses (nickel eyeglass dermatitis)

may be applied by simply impregnating a small linen square with the suspect material and proceeding as outlined.

The results of these tests become significant only when they are corrobor-

rated by the history by the localization, appearance, and course of the eruption, and by the effects of the elimination of and subsequent reexposure to the suspected agent.

In many instances the history and the appearance and localization of the



FIG. 162 Chemical Contact Dermatitis. Is better due to mercury in shampoo lotion. This dermatitis improved rapidly following application of aluminum water several times a day.

eruption will at once lead to the discovery of the etiologic agent.

Sites and Agents Following are some of the more or less characteristic sites of contact type dermatitis and their respective most common causes.

SCALP Usually caused by scalp lotions, scalp tonics, pomades, hair dyes, hair acids, etc. The allergen may be karaya gum, chemical used in cold wave permanents (thioglycolic acid) or hair lacquers.

FOREHEAD The causal agent may be bathhands, hat linings and other material of hats.

EYELIDS Numerous substances may be responsible such as cosmetics, soaps, hand lotions, creams, face powders, nail

polishes and lacquers, mascara, eyebrow pencils, eyelash dyes, etc. Air-borne, volatile agents and dusts (insect sprays, gaseous substances, nasal sprays, cleaning fluids, ant moth preparations, perfumes, benzene; karaya gum (found in some tooth pastes, denture adhesive powders, some candies such as jelly beans, some laxatives, and some salad dressings) dusts from clothing furniture, air borne pollens, etc., materials of dyed clothing, fabrics, furs, gloves, handbags, etc.)

FACE IN GENERAL The cause may be face powders, creams, rouges, lotions, all possible materials transferred by hands or air borne. All substances used on the face scalp, or hands. Shaving soaps, after shaving lotions, etc.

LOOSES OF EARS Earrings, bakelite other plastics, nickel, white gold other metals, etc.

RETROAURICULAR AREA AND BRIDGE OF NOSE Spectacle frames.



FIG. 163 Chemical Contact Dermatitis. Due to soap powder.

NOSE AND NASOLABIAL AREAS Nose drops, nasal ointments, sprays, etc., perfumes, handkerchiefs, paper tissues, etc.

LIPS AND PERIORAL AREAS Lipsticks and their dyes, mouthwashes, tooth pastes, powders. Sometimes certain

foods such as oranges, and other citrus fruits

SIDES OF NECK, UPPER CHEST UPPER BACK PERIAXILLARY AREAS ANTECUBITAL SPACES (Typical symmetrical distribution usually occurring in middle-aged women and often associated with obesity)



Fig. 164 N. skin Dermatitis.

and hyperhidrosis). Dress materials and their dyes (any color but black dark blue and dark brown are most common offenders) often seen in middle-aged women first going into mourning

NECK (FRONT SIDES AND BACK) Collars, scarfs, neckties, furs, fur dyes, substances used in scalp, necklaces, perfume (*berlock dermatitis*), wave sets, hair cosmetics and dyes, hairpins, etc. nail lacquers, cosmetics and other substances used on face, creams, etc.

AXILLARY AND PERIAXILLARY AREAS Antiperspirants, deodorants, depilatories, dress shields, dyed materials, perfumes, shaving materials

HANDS FOREARMS AND FACE Substances too numerous to list. Most occupational and industrial excitants. Sub-

stances encountered in hobbies, games, avocations, professions; soaps, cleansers, plants, gloves, steering wheels, instruments, rings, bracelets, cosmetics, topical medicaments, applied to self or to others all objects which may be touched, handled held or worn. Eczematoid dermatoses of the hands are common. Itching is almost always present and the original condition often distorted by the rubbing and scratching. The lesions are often chronic, persistent relapsing or recurrent. The etiologic factors are varied and often obscure. Contact appears to be the commonest agent but ringworm, *ids* (bacterids and dermatophytids), pompholyx, direct staphylococci or strepto-



Fig. 165 Dermatitis Venenata Showing considerable edema of neck due to finishing material in shorts. (Courtesy of Dr. Carroll S. Wright.)

cocci infections, epidermodermatitis (Gougerot), infectious eczematoid dermatitis (Engman) and a frictional dermatitis may be primary or secondary factors also. All of these may be clinically indistinguishable.

TRUNK. Clothing, brassieres, girdles, underwear night clothes, sweaters, bathing materials, bath salts, soaps, perfumes, massage creams, etc.

PERIANAL. Suppositories, douches, sub-stances in enemas, intestinal parasites, ingested foods (fruits, oils) topical me-



Fig. 166 Contact Dermatitis. With secondary infection. Four months duration. Primary excoriation on toilet-seat.

dicaments, underdrawers, sanitary napkins, toilet paper

VULVAR AND PERIVULVAR. Douches, anticonceptual jellies, suppositories, sanitary napkins, etc. Substances carried by the hands, perfumes, deodorants, prophylactic agents, etc. condoms, pessaries, etc.

PENIS AND SCROTUM. Condoms, prophylactic agents, anticonceptual medications, douches (used by partner) etc. colored drawers, rubber and elastic supporters, substances carried by the hands, plant (poison ivy etc.)

THIGHS, LEGS AND ANKLES. Dye materials and materials of trousers, under-

drawers, socks, etc. match boxes, cigarette lighters, coins, and other metallic objects carried in trouser pockets, etc., volatile and air borne substances, dusts inside trousers, etc. Garters (rubber elastic dyes, metal clasps)

LOWER PORTIONS OF LEGS AND FEET. Shoes, socks, stockings (leather dyes, tanning agents, dyes, and finishers materials, etc.)

FEET (Particularly dorsum of great toes, sides, and dorsum of feet and sometimes soles—often with little interdigital involvement) Shoes, leather dyes, tanning agents, shoe polishes socks and their dyes, and finishers, rubbers, etc.

MOHS OR LEGS GENERALIZED ERUPTIONS. Any of the aforementioned agents may



Fig. 167 Chemical Contact Dermatitis. Due to "box-over" used to mend "runs" in hose.

produce not only localized but general-ized eczematous dermatitis. Also, medications applied to numerous or wide-spread areas, and eczematogenous medications taken by mouth or injected (arsenicals, quinine, salicylates, etc.)

The two most important groups of causes of eczematous dermatitis are (1) prescribed medicaments or proprietary remedies used in local treatment and (2) occupational and industrial substances. The substances in both of these groups are most likely to cause eruptions at the site of actual application or most direct exposure. But both groups may cause disseminated eruptions in areas distant from the sites of direct exposure and they can also cause generalized dermatitis. The dissemination of an excitant usually takes place by external distribution but may occur by diffusion or via the lymphatics or blood stream. In cases of external dissemination the hands, face and genitalia are often affected.

Nonspecific Therapy This consists in the injection of protein material which has no direct connection with the disease in question. It is carried out by many substances; usually readily available. One of the simplest measures is auto-hemic injections consisting of withdrawal of 10 cc. of blood from a vein and immediate injection into the buttock. Milk and milk products are also used. Daily intravenous injections of 1 gm. (15 grains) of sodium thiosulfate or of calcium chloride are occasionally of value.

Local Treatment This depends on the stage of the eruption. The mildest of all applications is the *wet dressing* which is used in the vesicular and exudative stages, it relieves the pruritus. The common wet dressings are milk, boric acid solution (3 per cent), liquor aluminum acetate (1 per cent) (probably the best), tannic acid (2 per cent), aqueous solution silver nitrate (0.25 per cent) and potassium permanganate (1:10,000). Wet dressings should be renewed every three hours. During the night, *calamine liniment* may be substi-

tuted since wet dressings must be changed frequently which would interfere with the patient's sleep.

An excellent lotion is

Liq. phenol	48
Glycerin	100
Kaolin	410
Aluminum phosphate gel (N.N.B.)	
q.s. ad	2100
Sig. Appl. frequently	

X-ray therapy in small fractional doses, aids in relieving pruritus and promoting resolution of the lichenified areas.

When the vesicles dry up and scaling appears, a mild ointment such as menthol phenol paste or ichthyol zinc paste should be used. This may be applied on old muslin (by means of a wooden tongue blade) about as thickly as butter on bread which in turn is applied to the lesions with the ointment side to the skin. The dressing is changed morning and night and the ointment remaining on the lesion is removed gently with mineral oil, after which a fresh dressing is applied. Water and especially soap, should be avoided.

Patients with extensive eruptions should be hospitalized; institutional care will provide more comfort and better treatment. A colloid bath may be given once or twice daily to be followed with the applications mentioned depending on the stage of the eruption. In sub-acute eruptions, the lesions may be thickly covered with a paste of borated cold cream, zinc menthol phenol, or ichthyol zinc.

After contact dermatitis has become chronic, stronger applications must be used but not before having tested the sensitivity of the skin by means of a mild preparation such as ichthyol zinc paste or menthol phenol paste. After a few days, in case there is no irritation *pine tar* and *salicylic acid ointment* may

be prescribed, starting with 0.5 per cent of each, and gradually increasing the strength of each up to 5 per cent. Remember always that dermatoses are not infrequently made worse and prolonged by the applications designed to cure them. The superadded cutaneous reactions are the result of applications which are too strong (direct irritants to a skin already immunologically weak) or of too intensive a therapy or of chemicals to which the skin is already sensitive.

After the tests have been made, and the offending substance has been discovered, the use of a protective covering will often suffice to prevent recurrence of the dermatitis. The various substances consist of greasy or soapy compounds designed to form a film over the skin for protection. Application of the material should be made before exposure, and after the day's work the exposed regions should be thoroughly washed with soap and water. A reliable preparation for protection of the skin is lanolin or aquaphor. In instances where scratching is prolonging the disease, daily bandaging of the parts by the attendant or physician himself has a psychologic effect of therapeutic value.

In chronic dermatitis venenata of the forearms, hands and feet desensitization may sometimes be obtained by the cataphoretic method of Lowenfish. The medium used is tap water placed in two glass trays (12 by 7 by 3 inches). In each tray is placed a 4-inch square thin copper plate to one of which is attached the negative and to the other the positive pole. The affected hands are placed one in each tray and the current turned on, first to 5 m.a. and then slowly up

to 15 or 20 m.a. or a point just below a tingling sensation. The treatment is continued for twenty to thirty minutes, one to three hours weekly. Good results should be obtained in most cases in six weeks or less.

Prophylaxis. Prophylactic therapy in the case of hypersensitiveness to plants may be carried out by annual injection of plant extract, which should be made four to six weeks (or earlier) before the patient contacts the irritant. In the case of poison ivy extracts, directions vary with materials supplied by various firms, but usually two injections are given spaced two weeks apart. The consensus is that these extracts should not be used to treat acute ivy dermatitis; and if used, only small daily doses should be given, such as have been advised by French and Halpern: 0.1 cc. of a 1:10 dilution of a 5 per cent alcoholic extract of poison ivy leaves; the dose may be increased to 0.4 cc. by the fourth day.

A cream for water proofing the skin when there is prolonged contact with water is as follows (P.R.B. III)

White wax	100
Hydrogenated wool fat	25
Sulfonated olive oil	500
Petroleum, q. ad	1000

A number of protective and water resistant compounds for prophylactic use against industrial dermatoses are on the market (such as Breck's cream or paraprof [National Drug Co.])

The following is protective and water repellent

Shelloc	10.0
Talcum	10.0
Tetralin oxide	10.0
Isopropyl alcohol	100.0

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tuted since wet dressings must be changed frequently which would interfere with the patient's sleep.

An excellent lotion is

Liq. phenol	33
Glycerin	100
Kaolin	250
Aluminum phosphate gel (N. R.)	
q.s. d	2100
Sig. Apply frequently	

X ray therapy in small fractional doses aids in relieving pruritus and promoting resolution of the lichenified areas.

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cytostoma duodenale. In such cases, the ova or parasites are present in the stool within six weeks after the cutaneous invasion, commonly present on the feet or legs and especially between and beneath the toes.

Prognosis The prognosis is good.

Prophylaxis The wearing of shoes or sandals while walking on infested beaches or on damp cellar floors is a prophylactic measure.

Treatment The larvae can occasionally be located beyond the terminal ends of burrows by applying a drop of ordinary lubricating oil to the skin. This oil makes the skin sufficiently translucent so that the larvae can then be seen with a binocular microscope. Larvae appear as small black dots. They can be removed by a sharp needle.

Spraying the end of a burrow with

ethyl chloride for a period of two or four minutes produces favorable results. Solid carbon dioxide applied to burrows is equally effective.

Carbon dioxide is painful and produces slow healing. Intramuscular injections of antimony compounds have been occasionally successful when the above methods fail. Smith advises 2 cc. of a 0.3 per cent solution of *fusidin* once daily for five days with a repetition of the course of injection one week later.

The following has been advised for the pruritus:

Boric Acid	4.5
Thymol	0.1
Methyl salicylate	0.2
Oil of peppermint	0.1
Oil of camphor	0.0
Oil of eucalyptus	2.0
Essence	50.5
Glycerin	37.5

CRYPTOCOCCOSIS EPIDERMICA

SYNONYM *Stomatomyces epidermici*.

This is a dermatosis caused by the *Cryptococcus parvulus*.

Symptoms The sites of predilection are the eyelids and bends of the elbow. The lesions are café-au-lait in color and are macular although some may be slightly infiltrated. Furfuraceous scaling occurs which resembles that of pity-

riasis *versicolor*. The causative organism may be seen on microscopic examination of the scales.

Treatment The treatment consists of the use of antiparasitic ointments containing 3 to 5 per cent precipitated sulfur, 3 per cent ammoniated mercury or 1 per cent betanaphthol.

CUTANEOUS DIPHThERIA

Etiology The Klebs-Loeffler bacillus (*Corynebacterium diphtheriae*) may produce primary or secondary cutaneous disease. During World War II, small epidemics of cutaneous diphtheria have occurred in the military personnel in all of the warm-climate theaters of operation. Primary diphtheria of the skin is generally contracted through direct contact with one who is a carrier or who

has the disease, or through fomites (contaminated dressings, clothes, and the like). Most reported cases, however, have been the result of complicating automuculation with the organism from diphtheritic rhinitis or from faucial diphtheria. Owing to the general use of antitoxin, this type of cutaneous diphtheria has become rare. It is assumed that some break in the continuity of the skin

CREEPING ERUPTION

SYNONYMS: *Larva migrans*, *myiasis linearis*, *sandworm disease*

Creeping eruption identifies a variety of skin lesions produced by burrowing larvae and characterized by tortuosity and migration.

Varieties and Etiology The majority of cases in America are due to infestation by the larvae of cat and dog hookworm (*Ancylostoma braziliense*). Contact with damp sand and earth contaminated by excreta of dogs and cats is the usual mode of infection. A similar dermatitis is produced by the larvae of the *Ancylostoma caninum* which infects dogs and cats. Cases of larva migrans in the Orient are often due to pig and cat nematodes of the genus *Gnathostoma*. The larvae of the horse bot fly (*Catrophilus*) have also been known to produce larva migrans.

The larva (*Ancylostoma braziliense* or *Brazilian hookworm*) is approximately $\frac{1}{10}$ of an inch in diameter and $\frac{1}{20}$ of an inch long. The motile filariform larvae burrow under the stratum corneum.

Incidence Creeping eruption occurs all over the world especially in Russia. Children and young adults are more often affected than the aged.

Pathogenesis Microscopic examination shows the larvae in the various layers of the epidermis. The larvae of the horse bot fly (*Catrophilus*) occurs more superficially than the other larvae and it also produces less inflammatory reaction than do those of the Nematodes.

Symptoms The onset of creeping eruption is characterized by slight local itching due to migration of larvae within their burrows and the appearance of papules at sites of the larvae location. Intermittent stinging pain is common. Red tortuous lines are formed in the skin. Migration of the larvae occurs

about four days after infection and progresses 1 or 2 inches each day. Linear lesions are often interrupted by papules which mark the site of larvae. The line assumes an irregular and tortuous course which broadens as it advances. The distal advancing end is more inflamed than



Fig 168 Larva migrans.

the proximal end. The length of an erythematous line depends upon the duration of infection and the speed of larval migration.

Diagnosis The limited location of the lesions, together with the history and picture of its development is diagnostic. The length and tortuosity of the burrows distinguishes creeping eruption from scabies. Occasionally creeping eruption must be differentiated from papulovesicular dermatitis (ground itch or hookworm dermatitis) caused by the larvae of the *Necator americanus* and/or *An-*

CUTANEOUS HORN

SYNONYM *Corn cutaneum.*

Cutaneous horns are circumscribed conical or cylindrical shaped horny growths arising from the skin.

Incidence Both sexes are equally affected. It usually occurs after middle life although a case has been observed in infancy.



Fig. 170. Giant Cutaneous Horn.

Etiology The exact cause is unknown. Persons exposed to wind and strong sunlight are more apt to develop this condition if they are blonds and have a poorly pigmented skin. According to Montgomery (Douglas) the so-called cutaneous horn may be a group of unusually coherent epithelial cells springing up from a circumscribed base, in which epitheliomatous degeneration often occurs, or from the bowl of a sebacous cyst, or it may be a highly keratinized papilloma or wart with unusually elongated papillae. It may also be one of the numerous malformations found in

Pathology: Histologically cutaneous horns are closely related to verrucae. The diagnostic histological finding is the marked hypertrophy of the horny layer and this is out of all proportion to the other layers. Small areas of parakeratosis are present. The stratum mucosum is also hypertrophied. The rete processes are similar to those seen in keratosis wulfia. The dermis shows elongation of the papillae. Horny pearls are seen in the rete. Eventually malignant degeneration usually occurs.

Symptoms This affection develops slowly and usually consists of a single



Fig. 171. Filiform Cutaneous Horn. Of the right cheek.

lesion which is yellowish or brown to black in color. It begins as a warty growth and when fully developed it resembles the horn of lower animals. The base is usually erythematous and elevated from the normal plane of the

precedes the infection: Secondary diphtheritic infection is seen in wounds (insect and leech bites and minor abrasions in particular)

Symptoms Primary cutaneous diphtheria assumes a variety of acute and



Fig. 169: Cutaneous Diphtheria. Primary diphtheria of the skin of the face. Note crusted eczematiform lesion on right and left cheeks, buccal commissures, and labionasal junctions. Cultures positive for Klebs-Loeffler bacillus. Three thousand units of diphtheria antitoxin resulted in almost complete disappearance of lesions in thirty-six hours.

chronic localized and generalized clinical forms. These are vesicles, bullae, impetigo, ecthyma, ulcers, gangrene and pustular and eczematous dermatitis. One or more lesions may be present. Constitutional symptoms may or may not be manifested and in occasional cases

develop only some time after the lesions have persisted. Toxic neuritis and myocarditis occur. In those with nasal or oral diphtheria the cutaneous lesions are apt to develop about the nose and mouth and other body orifices (vulva, anus, ears) and are apt to be dry and eczematous. When a wound is secondarily infected with the diphtheria bacillus, local symptoms of inflammation are increased and a soft grayish fibrinopurulent crust generally develops. The underlying ulceration is punched out and rounded with a base that is clean or covered with a gray exudate. The borders of the wound are well defined and edematous. The draining lymph nodes become enlarged. Veldt or desert sore is considered in at least some instances a type of cutaneous diphtheria (see p. 245).

Diagnosis and Treatment Clinically there are no special objective signs upon which a diagnosis of cutaneous diphtheria can be made. One's suspicions should be aroused in the presence of cutaneous lesions in those with nasal or oral diphtheria, especially in the ill child with a chronic nasal discharge. Finding the organism in smears and cultures from the lesion is suggestive. Virulence tests may be required to rule out pseudocutaneous diphtheria. The rapid disappearance of these organisms simultaneously with healing of the skin lesions following intramuscular injections of diphtheria antitoxin (20,000 to 60,000 units) is proof positive of a primary infection. When diphtheria organisms are secondary invaders, they tend to disappear from the lesions under compresses soaked in penicillin solution (1000 units per cubic centimeter of isotonic salt solution).

on the inherent or acquired sensitiveness of the skin to mechanical trauma, vary in appearance from an acute, rapidly disappearing inflammation, when trauma is stopped, to a chronic, thickened skin, with scurf or crusts and erosions, all of which disappears slowly when trauma is eliminated. A frictional dermatitis of varying degree is a superimposed state in all pruritic dermatoses, irrespective of their origin and sensitivity. Thus, in a dermatitis venenata or a contact dermatitis with pruritus, the only sign of intolerance, which the effects of rubbing and scratching may produce, is a mild frictional dermatitis; but in those with severe chemical dermatitis, the superimposed frictional dermatitis may add much to the severity and duration of the dermatosis. These secondary frictional dermatitides are well known and often, in addition, secondarily infected. In pure or primary psychogenic pruritus, a frictional dermatitis of varying severity and extent is also observed. In some only small areas may be involved, in others the eruption may be very extensive.

The character of the patient's skin appears to play a considerable part in the objective results produced by the rubbing and scratching. Acute and chronic forms of frictional dermatitis result. The skin is reddened and eroded, oozing is present here and there, and it is covered with scales and crusts, but there are no vesicles or blebs such as are seen in eczema or dermatitis venenata. Habitus pruriticus is a special form of psychogenic pruritus.

In those forms related to sexuality the itching and scratching serve to produce perverted sexual gratification. In other types, resentment against members of the family, other people or their work, exhibitionism need for self-accusation and self-punishment appear to be important factors. States of frustrated and repressed

longing appear to be basic emotional conflicts in other patients. In any case, it is important to recognize and establish whenever possible just how much of a given dermatitis is frictional (all, or only part?) because it gives the observer a better idea of the proper therapeutic approach (see p. 636).

Urticaria Urticaria on a psychogenic basis, is generally accepted. In Wright's analysis of twenty-five cases of chronic urticaria, there was a history in seventeen of them, of some definite shock, or of worry or nervous exhaustion preceding or accompanying the onset of the illness. This corroborates the percentage of psychoneurogenic factors in urticaria reported by Stokes, Kulchar and Pillsbury. In addition, numerous reports of psychogenic urticaria are quoted by Dunbar, Becker and Obermayer have pointed out that the threshold of an allergic response may be raised or lowered at a given time by the states of one's emotional tension. Thus, a patient with a food urticaria may be able to eat a food to which he is hypersensitive when he is free from tension or anxiety. Dunbar has shown that symptoms may be eliminated by psychotherapy while the cutaneous reactions to the specific substances remain unchanged (see p. 806).

Cutaneous Hyperesthesia This is symptomatic of local or systemic disease. It is a symptom-complex rather than a disease entity. Complete or partial hyperalgesia, anesthesia, and paresthesia are cutaneous manifestations of some disturbance in the cerebrospinal system or within peripheral nerves.

Dermatalgia This is neuralgia of the skin. It is characterized by painful cutaneous sensations without evident causes. Dermatalgia may be accompanied by cutaneous hyperesthesia and by painful sensations in structures un-

quent to mental shock and emotional conflicts of various kinds and it has been stated that erythemas, ecchymoses and blebs have been produced upon anaesthetic areas induced in the skin by suggestion while the patient was under hypnosis.

In a different sense, but certainly related to the psychologic processes, is the erotic pleasure produced in many people by simple stroking of the skin. In this latter instance, however, the skin rather than the brain is the primary receptive organ. In some of these cases, the urge which is clearly erogenous, is carried to the point of furious rubbing and scratching with the production of a thickened fissured skin showing frictionally produced eroded encrusted surface lesions. I have observed on a physician's right thigh an area larger than a man's palm of chronic thickened frictional dermatitis of many years duration. He obtained complete mental and physical relaxation, and a good night's sleep by thoroughly rubbing the area at bedtime, when the itch sense became urgent. Many cases of pruritus ani, scroti, perinei and vulvae are of this category, i.e. on an erogenous basis. They may however be part of a purely psychogenic state, based upon emotional conflicts such as fear of pregnancy or repressed hostility against a husband philanderer. In some of these latter cases, spinal analgesia may be of diagnostic value. It is important for the observer to remember that in all cutaneous neuroses the subjective symptoms are never imaginary and the patient's symptoms are real to them. The itching sense however presumably originates centrally rather than peripherally (skin) as it does in scabies or pediculosis. When pruritus is the chief complaint, the best way to make such a patient's condition worse or at least to upset him emotion-

ally is to tell him that he does not itch and that it is all "nerves."

The symptoms in psychogenic dermatoses are local or general. They may be purely subjective, objective or both, as in dermatitis factitia (see p 260) or other self-induced eruptions, including frictional dermatitis (due to rubbing and scratching) and emotional dermatitis (see p 220).

They may be (1) primary, i.e. develop without a preceding cutaneous ailment and be purely psychogenic from the onset or (2) secondary, i.e. follow a non-psychogenic cutaneous ailment. These latter types are usually of short duration but may be prolonged. Thus, the development of an acute dermatitis venenata may be the initiating factor leading a patient with a "dormant" psychoneurosis to his mental conflicts to and against the skin. In such cases, the cutaneous condition is indefinitely prolonged as a psychogenic (objectively frictional) dermatosis. An unusually large number of eczematous dermatoses are thus prolonged and unless the psychogenous element is recognized the condition becomes chronic.

Pruritus. Primary psychogenic pruritus (local or generalized) with objective skin disturbances (frictional dermatitis) is a well recognized state by most dermatologists. Some factors have already been enumerated.

Frictional dermatitis (objective sign of neurodermatitis) may when viewed as psychogenic be primary or secondary. The primary types are (1) initiated by or accompanying psychoneurotic states or (2) on a pruritic basis of organic origin. The secondary types are those superimposed upon an existing endogenous, allergic, atopic or contact dermatitis. A dermatitis due to the mechanical action of rubbing or scratching may depending

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for parasitophobia and certain special phobias, acarophobia and pediculophobia.

Acarophobia. Although this term means fear of the acarus it really refers to a fear of already having scabies. It commonly occurs in those recently treated for this disease. A similar state is seen in pediculophobia usually in connection with pubic lice. Itching is complained of and, in one case reported by Wright, had persisted for years.

Parasitophobia. This is a psychic state or false belief regarding his body in which the patient erroneously believes his skin to be infested with some parasite usually insects or fleas, to which he attributes the itching. Patients affected with this disorder pick small pieces of epithelium and other debris from the skin and often bring them for examination with the insistence that the offending parasite which they say is contained within this debris, be submitted for investigation.

This condition may occur in the course of a number of grave mental abnormalities such as dementia praecox, toxic psychoses, involutional melancholia, paranoia and paranoid states. It has been observed in psychoneurotics and obsession-compulsion states, although in the latter it is not a true delusion since they have sufficient insight to realize that the condition does not actually exist.

Rhynchophobia. This is a psychic disorder in which there is a fear of bakiness. An actual progressive alopecia may or may not be present.

Cancerophobia. This neurosis is becoming increasingly more frequent. It may originate in actual or imagined mucocutaneous lesions. It is commonest in relation to the mouth and lips. More than half of the patients with stomatopyrosis or glossodynia have it. In most instances only careful questioning will elicit the fear. If the physician makes an "intui-

tive" diagnosis before the patient even admits it and simply states the fact that no cancer is present, the sigh of relief and the joyful facial expression confirm the diagnosis. Nevil and other cutaneous le-



Fig. 178. Nervous Excoriations.
(Courtesy Dr. J. V. Klander.)

stions may be the cause for its development, in many cases it develops after the patient has read of, heard of or seen people with cancer. Reassurance usually cures.

derlying the skin. The disease is preceded by some obscure disturbance in the nerve centers or within the course of peripheral nerves.

Meralgia Paraesthetica (*Crurum Paraesthetica*, Restless Legs, Leg Jitters) This is paresthesia accompanied by persistent tingling numbness, and other abnormal cutaneous reactions on the outer aspects of the legs and thighs, often when the patient is still. There is a feeling of weakness in the legs and a sensation of cold in the feet. Functional or organic disease of the nervous system, prostatic disease as well as testosterone deficiency have been suggested as the cause (Winter).

Syphilophobia This is abnormal fear by the patient that he is afflicted with syphilis. It is the common expression of anxiety concerning some sexual act and is amenable to treatment by psychotherapy.

Bromidrosiphobia This is a neurosis in which the patient is convinced that his sweat is offensive. The phobia like other neuroses may be the early manifestation of dementia praecox.

Neurotic Excoriations Cutaneous excoriation occurs as a result of mechanical trauma among some individuals who habitually subconsciously and even deliberately pick themselves. The lesions follow repeated picking, digging and scraping parts of the body readily accessible to the hands. The habit is commonly subconscious and is developed as a result of the belief that it corrects some cutaneous abnormality. The lesions are linear and may be superficial or deep depending upon the extent and duration of trauma. In some cases, the patients pick at actual lesions on the skin. This is common in acne vulgaris (excoriated acne of young people). Many cases of acne are made worse by this habit. It

appears, in all cases, to be a compulsive neurosis.

Trichotillomania This is a neurosis characterized by an abnormal desire to pull out the hair. A mild pruritus of the scalp or of the involved hairy parts is an accompanying feature. The sites of



Fig. 172. Neurotic Excoriations. These are usually located on the face.

involvement are the frontal region of the scalp, the eyebrows, and beard.

Trichokryptomania This is a similar disease in which the hair is broken off instead of being pulled out.

Dermatothalasia This is a cutaneous neurosis characterized by an uncontrollable desire to rub or scratch one or more areas of the skin.

Delusion of Parasitosis This term, suggested by Wilson and Miller, is used

Etiology It is considered a *vasomotor neurosis*. It is common in the adult male and often worse in summer. It may be symptomatic of functional or inorganic central or peripheral disease of the nervous system, such as lumbar sclerosis, tabes dorsalis, paralysis, and myelitis. McLeod has found sclerotic and obliterative changes in the smaller blood vessels.

Diagnosis It should be differentiated from Raynaud's disease in which the his-

tory is different, in which remissions occur the age incidence is lower and in which according to Mitchell, there is a livid and not a purplish redness.

Treatment *Rest* in the recumbent position, *elevation* of the parts, and *cold compresses* relieve the burning pain, swelling, and redness. Hypodermic injections of *epinephrine* ($\frac{1}{2}$ cc. of 1:500 solution in oil once daily) are beneficial. *Acetylsalicylic acid* and *phenobarbital* have been recommended.

Treatment There is no short cut although many of these patients eventually cure themselves. It is important to establish their confidence in you on the first visit. A return to emotional stability is an essential for success. This is best achieved by a sympathetic patient attitude on the part of the observer. One



Fig. 174: Neurotic Excoriations.
(Courtesy of Dr. J. V. Kunder)

must listen carefully and with interest to the patient's mental woes as they relate to the personal, social and work environment. The patient must be allowed and even urged to talk freely without fear or condemnation. Patients feel relieved and will then often cooperate. In this way also they may lose their sense of guilt, self-blame or hostility and begin to understand that the skin condition is only the expression of the underlying emotional conflicts. However, patients are not always able to put into practice the objective viewpoint or insight gained through such mental catharsis. Failure is certain without the patient's cooperation, but persistence often succeeds since time appears to be the important factor

In some cases of supposed chronic dermatitis venenata the psychic element may require careful evaluation, since it may be the major etiologic factor. All of these patients, whether the cutaneous condition is localized or generalized should receive a thorough physical examination, including serologic and blood studies. They should be constantly impressed with the fact that the viscera are normal. A remark that an organ is normal helps the patient's mental state considerably. *Reassurance* in some and *rationalization* in all offer the only reasonable approach to permanent cure. Local therapy is incidental although necessary. *Hypnosis*, *pentothal sodium interviews*, *narcosynthesis* and *electric shock therapy* have been of value in some selected cases. Some patients are temporarily even permanently cured by *minor operations* of one sort or another (see pp. 630 and 631 for special therapy). *Sedatives* should be used as temporary, not permanent therapeutic agents. *Change of environment or occupation* is occasionally necessary. Finally, these dermatoses are the patient's solution of an emotional conflict and some are best left untreated.

Erythromelalgia

SYNONYMS *Wells-Mitchell disease*, *red neuralgia*.

This is a rare syndrome characterized essentially by (1) attacks of unilateral or bilateral painful or throbbing swelling of an extremity with or without sensory disturbances, (2) the appearance of vasodilatation of the affected part as shown by redness or purplish redness, pain and elevation of the local temperature. Heat and dependency of the parts aggravate or initiate this condition. The attack persists for several days. The disease has a slow progress and may lead eventually to gangrene.

Etiology It is considered a vasomotor neurosis. It is common in the adult male and often worse in summer. It may be symptomatic of functional or organic central or peripheral disease of the nervous system, such as insular sclerosis, tabes dorsalis, paralysis, and myelitis. McLeod has found sclerotic and obliterative changes in the smaller blood vessels.

Diagnosis It should be differentiated from Raynaud's disease in which the his-

tory is different, in which remissions occur the age incidence is lower and in which according to Mitchell, there is a livid and not a purplish redness.

Treatment Rest in the recumbent position, elevation of the parts, and cold compresses relieve the burning pain, swelling, and redness. Hypodermic injections of *epinephrine* ($\frac{1}{2}$ cc of 1:500 solution in oil once daily) are beneficial. *Acetylsalicylic acid* and *phenobarbital* have been recommended.

DECUBITUS

SYNONYMS: *Bedsore trophic ulcer pressure sore*

Decubitus is local ulceration and necrosis of the skin and subcutaneous tissue

Etiology: Continued pressure exceeding 1.5 pounds per square inch of skin over bony prominences causes partial and complete occlusion of the arterial blood supply which leads to necrosis and gangrene. Lesions of the central and peripheral nervous system can interfere with the blood supply thus leading to decubitus. Trophic ulcers occur following injuries to the spinal cord and paralysis. Debilitating diseases like tuberculosis, cancer and typhoid fever are often accompanied by bedsores. Chronic irritation of the skin from fecal matter, urine, and skin secretions, plus secondary infection play a part in its development.

Symptoms: The first symptom is that of erythema following this stage, the area becomes macerated and finally ulcerated. The sites of predilection are areas of bony prominences like the sacrum, coccyx, scapulae, elbows, heels, and trochanters. Pain is usually present unless decubitus is due to some lesion in the nervous system.

Prophylaxis: Cleanliness and the proper care of the skin free use of zinc bismuth powder and rubs with camphorated oil are prophylactic measures. Pressure on exposed parts should be relieved by frequent changes of the pa-

tient's position, air cushions, and a water mattress. Avoid creased bed linen.

Treatment: Lead and opium wash (lotio plumbi et opii) compresses and 5 per cent tannic acid sprays are useful in the preulcerative stage. Applications of heated Unna's paste are commended in the presence of ulceration. The following preparation has given remarkable success in the Philadelphia General Hospital.

Menthol	10
Acid salicylic	120
Zinc oxide	150
Amyl	150
Petrolat. q. s.	2000

This is a protective, antiphlogistic, and healing ointment. Others are

I	
Silver nitrate	05
Balsam Peru	10
Petrolatum	300
Sig. Apply twice daily	

II	
Zinc sulfate	25
Lead acetat.	50
T. myrrh	10
Petrolatum q. s.	500
Sig. Apply daily	

Ten per cent anesthesin (ethyl aminobenzoate or benzocaine) powder may be added to either of the latter ointments if pain is present.

In severe decubitus, a plasma protein concentration study is indicated if necessary blood plasma should be used.

DERMATITIS

Dermatitis is an inflammation of the skin. It has the same pathology and characteristics as eczema. The term is, however, reserved for those cutaneous

inflammations which result from the action of certain factors to which the skin of the individual patient possesses an idiosyncrasy.



Fig. 175 *Dermatites Enfoliata*. (Courtesy of Dr. Carroll S. Wright.)

DECUBITUS

SYNONYMS: *Bedsore trophic ulcer pressure sore*

Decubitus is local ulceration and necrosis of the skin and subcutaneous tissue

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Symptoms The first symptom is that of erythema; following this stage the area becomes macerated and finally ulcerated. The sites of predilection are areas of bony prominences like the sacrum, coccyx, scapulae, elbows, heels, and trochanters. Pain is usually present unless decubitus is due to some lesion in the nervous system.

Prophylaxis Cleanliness and the proper care of the skin, free use of zinc, bismuth powder, and rubs with camphorated oil are prophylactic measures. Pressure on exposed parts should be relieved by frequent changes of the pa-

tient's position, air cushions, and a water mattress. Avoid creased bed linen.

Treatment *Lead and opium wash* (lotio plumbi et opii) compresses and 5 per cent *tannic acid sprays* are useful in the preulcerative stage. Applications of heated *Unna's paste* are commended in the presence of ulceration. The following preparation has given remarkable success in the Philadelphia General Hospital:

Menthol	30
Acid salicylic	120
Zinc oxide	150
Amyl	150
Petrolat. q.s.	2400

This is a protective antiphlogistic and healing ointment. Others are:

I	
Silver nitrate	30
Balsam Peru	10
Petrolatum	300
Sig. Apply twice daily	
II	
Zinc sulfat.	25
Lead acetat.	50
Tr. myrrh	10
P. trolatum q.s.	300
Sig. Apply daily	

Ten per cent *anesthestin* (ethyl ammonio-benzoate or benzocaine) powder may be added to either of the latter ointments if pain is present.

In severe decubitus, a plasma protein concentration study is indicated if necessary blood plasma should be used.

DERMATITIS

Dermatitis is an inflammation of the skin. It has the same pathology and characteristics as eczema. The term is, however, reserved for those cutaneous

inflammations which result from the action of certain factors to which the skin of the individual patient possesses an idiosyncrasy.

The cutaneous irritants which are causative of dermatitis designate the following varieties:

Dermatitis actinica (erythema solare)
 Dermatitis anabastensis
 Dermatitis atopica
 Dermatitis trophica
 Dermatitis actinotóxica
 Dermatitis colorica
 Dermatitis congelationis
 Dermatitis contact
 Dermatitis contactus/urinis
 Dermatitis, napkins
 (erythema of Jacquet)
 Dermatitis dysenterica
 (acutrocha)
 Dermatitis vasculosa infantum
 Dermatitis exuberans
 Dermatitis endotoxica neonata
 (BKier disease)
 Dermatitis gangrenosa infantum
 Dermatitis hemostatica
 Dermatitis herpetiformis
 Dermatitis kiemalis
 Dermatitis infectiva (infection
 cutanea atoides)
 Dermatitis maligna
 Dermatitis membranacea
 Dermatitis occupational
 Dermatitis papularis exulita
 (acne keloid)
 Dermatitis radium
 Dermatitis repens
 Dermatitis reactiva ray
 Dermatitis seborrheica
 Dermatitis toxica
 Dermatitis vegetans
 Dermatitis neonata

Dermatitis Actinica

SYNONYMS *Sunburn, erythema solare*

Dermatitis actinica is an inflammation of the skin produced by the actinic rays of the sun or by an artificial source, such as an ultraviolet lamp

Incidence. Blacks are more susceptible than brunettes. Small children tolerate only about one half of the exposure which produces symptoms in the adult

Dermatitis actinica is more common in the early spring. Exposure to the

reflected sun's rays from the snow on the surface of water or sand often causes severe reactions

Sensitivity to sunlight is also seen in porphyria, a rare familial inborn, metabolic fault. It may be (1) congenital, (2) acute, or (3) chronic. All three are really chronic diseases; but in the congenital

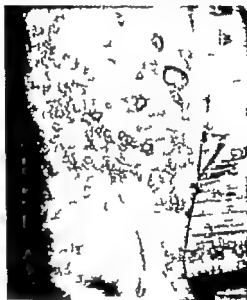


Fig. 176 Dermatitis Solare

and chronic forms, porphyria is excreted continuously. In acute toxic porphyria, however drugs of the sulphonmethane group have been incriminated. The urine and fecal porphyrins are increased in hepatic disease, especially in cirrhosis and toxic hepatitis from lead and alcoholism. Sensitivity to sunlight is also seen after the ingestion of the sulfonamides. Congenital porphyria is inherited as a mendelian recessive; acute porphyria is inherited as a mendelian dominant. Chronic porphyria includes those cases which exhibit the clinical symptoms of the congenital and acute forms. In the chronic form, the skin is sensitive to light, and vesicular-crusted skin lesions occur on

screen. The use of large hats and either red or black veils is also prophylactic.

The most effective preparations are those containing any one of the following remedies: quinine, tannic acid salol, methyl salicylate (10 per cent) benzyl salicylate (3 per cent) and the coumarines. By reason of pyoderma com



Fig. 178 Dermatitis Solaris. Patient while in darkness stupor was exposed to the sun with above result.

plecting sunburn, it is better to avoid oily and greasy antisunburn remedies and prophylactic preparations. Lotions and alcoholic suspensions tend to prevent the development of pyoderma and they are, therefore, preferable medicaments. Insoluble pigments in face powder are also preventive. Salsberger and Wolf recommend either of the following formulae

I	
Tannic acid	20
Salol	20
Spts. 100 rect. q. a. ad	1000
Sec. Apply before exposure to sun.	

II	
Methyl salicylate	11.5
Spts. 100 rect	100
Glycerin	3.0
Aqua	72.53
Perfume q	

M. Dissolve methyl salicylate in alcohol, add perfume glycerin, and stir filter.
Sec. Apply for prevention of sunburn.

Sutton and Sutton recommend the application of a thick coating of zinc

oxide ointment on which talcum is freely powdered.

Andrews believes mineral oil to be the best vehicle and also speaks favorably of glycerol monostearate and cetyl alcohol as valuable emulsifying agents.

Treatment A hot solution of boric acid (1 teaspoonful to the pint of water) applied as a compress is often very soothing.

Cold applications of a solution of *aluminum acetate* (1 to 2 tablespoonsful to the pint of water) is equally pleasing to some patients.

The liberal use of *zinc stearate* as a dusting powder or a *menthol powder* such as the following is very soothing:

Menthol	8.10
Zinc oxide	300
Talc powder.	300

Sec. Apply freely

Painful and tense blisters should be opened and the denuded surface painted with a 2 per cent aqueous solution of *gentian violet*.

Ointments should not be applied until the skin peels and the acute inflammation has subsided. The following soothing cream is recommended.

Boric solution	40
Ung. aquaphor	300
M. Then add Lanolin paste	30.0
Sec. Apply twice daily	

Andrews recommends *ice cold compresses* of mineral oil pieces of gauze should be wrung out in cold mineral oil and laid on the affected parts and gently rubbed with cracked ice.

Ephelides

SYNONYMS *Freckles, ephelis.*

Freckles are minute, circumscribed, rounded, lenticular and irregular macules ranging from a pale café-au-lait to dark brown color.

the exposed surfaces. The urine is red dish brown and contains porphyrin. Toxic porphyrin is excluded by complete elimination of irritating drugs.

Etiology Dermatitis actinica is not a true burn but an inflammatory reaction to the ultraviolet rays of a wave length varying from 2800 to 3200 Å units.

Symptoms The symptoms arise from two to twelve hours after exposure and

After the disappearance of the acute symptoms, the edema and erythema decrease, the epidermis exfoliates, and the pigmentation of the skin increases.

Exposure of the eyes to sunlight may produce edema of the conjunctivae.

Diagnosis A history of exposure to the sun or ultraviolet light is usually obtainable and makes for a certain diagnosis.



Fig. 177: Dermatitis Solaris

vary from a simple erythema to the formation of edema and bullae. Itching and burning are the usual symptoms, although headache, anorexia and pyrexia are occasionally present. In the very extensive cases of sunburn symptoms of collapse and nervous irritation suggesting acute meningitis, may occur. The sites of predilection are the exposed surfaces of the body. The face and flexor surfaces of the arms and legs are more sensitive than the extensor surfaces of the extremities. Some authors believe that the reaction of the skin to ultraviolet light is increased by acid diets.

Complications Vitiligo telangiectasia, herpes simplex, and lupus erythematosus may follow sunburn. These complications are more likely to follow exposure to the reflected rays of the sun from water or snow. Secondary pyogenic infection occasionally occurs.

Prognosis The prognosis is good. Secondary pyogenic infection may prolong the course of the disease which usually lasts from a few days to a week.

Prophylaxis Initial exposures of the naked skin to actinic rays should be of short duration unless the skin is protected with a filtering agent or sun

screen. The use of large hats and either red or black veils is also prophylactic.

The most effective preparations are those containing any one of the following remedies: quinine, tannic acid, salol, methyl salicylate (10 per cent) benzyl salicylate (3 per cent) and the cor marina. By reason of pyoderma com



Fig. 178 Dermatitis Solaris. Patient while in drunken stupor was exposed to the sun. As above result.

plicating sunburn, it is better to avoid oily and greasy antismear remedies and prophylactic preparations. Lotions and alcoholic suspensions tend to prevent the development of pyoderma and they are therefore, preferable medications. Insoluble pigments in face powder are also preventive. Sahberger and Wolf recommend either of the following formulas

I	
Tannic acid	20
Salol	20
Apts. ad rect. q. ad	1000
Use. Apply before exposure to sun	

II	
Methyl salicylate	11.5
Apts. ad rect.	1000
Glycerin	50
Aqueous	72.25
Perfume q	

M. Dissolve methyl salicylate in alcohol, add perfume glycerin, ad water. Filter.
Use. Apply for prevention of sunburn.

Sutton and Sutton recommend the application of a thick coating of zinc

oxide ointment on which talcum is freely powdered.

Andrews believes mineral oil to be the best vehicle and also speaks favorably of glycerol monostearate and cetyl alcohol as valuable emulsifying agents.

Treatment A hot solution of boric acid (1 teaspoonful to the pint of water) applied as a compress is often very soothing.

Cold applications of a solution of aluminum acetate (1 to 2 tablespoonful to the pint of water) is equally pleasing to some patients.

The liberal use of zinc stearate as a dusting powder or a menthol powder such as the following is very soothing:

Menthol	4.10
Zinc oxide	30.0
Talc perfumed	30.0
Use. Apply freely	

Painful and tense blisters should be opened and the denuded surface painted with a 2 per cent aqueous solution of gentian violet.

Ointments should not be applied until the skin peels and the acute inflammation has subsided. The following soothing cream is recommended:

Borax solution	4.0
Ung. aquaphor	30.0
M. Then add Lanolin paste	30.0
Use. Apply twice daily	

Andrews recommends ice cold compresses of mineral oil pieces of gauze should be wrung out in cold mineral oil and laid on the affected parts and gently rubbed with cracked ice.

Ephelides

SYNONYMS *Freckles*, *ephellæ*.

Freckles are minute, circumscribed, rounded, lenticular and irregular macules ranging from a pale café-au-lait to dark brown color.

Incidence Blondes and red haired persons are more subject to freckles than dark-complexioned brunettes. Freckles commonly appear between the age of ten and twenty-one years tending to disappear during adult life. They are however observed at all ages and in both sexes.

Symptoms Freckles are discrete smooth spots of the same apparent texture and level of the surrounding skin. They are more or less abundant but may be sparsely distributed. Freckles exhibit a predilection for the face (particularly the bridge of the nose and malar eminences) dorsa of hands and forearms, and other parts of the body exposed to sunlight. They are occasionally seen on covered parts of the body such as the back, buttocks, and genitalia and in these areas are known as "cold freckles." "Cold freckles" are better classified as pigmented nevi which are in reality lentigo proper. The term "freckle" should be restricted to pigmented macules resulting from the reaction of the skin to actinic rays.

Freckles are not congenital but appear in early childhood on exposure to bright sunlight during the summer. They fade during the winter except in countries of great altitude like Switzerland where the winter sunlight is of high actinic ray content.

Lentigo (see p. 592) occurs as an early symptom of xeroderma pigmentosum in which there is a high susceptibility to the actinic rays.

Mackee says "Lentigo and diffuse pigmentations may occur to a troublesome degree in certain individuals, particularly brunettes, as a result of a few mild exposures to x ray—a dose well within that required to produce a reaction. In such instances, if the treatment is discontinued the freckles and diffuse

tanning will disappear in a few weeks.

Lentigo Senilis Lentigo senilis is an interesting condition appearing among elderly persons and among younger persons in whom the integument undergoes degenerative changes. Atrophic thinning of the layers of the skin, loss of elastic tissue and the hypofunction of the sebaceous glands lead to dry scaling. The superficial veins become more prominent in lentigo senilis. Overproduction and accumulation of pigment take place. Pigmented lesions similar in appearance to freckles, appear and show a predilection for the dorsa of the hands, wrists, forearms, face and neck. They vary in size and exhibit great persistency. The majority of cases of lentigo senilis remain in the phase of freckling just described, but in a certain percentage of patients, malignant change takes place in pigmented lesions which ultimately turn into epitheliomas. These epitheliomas appear multiply and present varying stages of development and histopathology.

Prophylaxis Prophylaxis is important in treating freckles. Persons susceptible to freckling are kept under cover by wearing a sunbonnet and gloves before exposure to the sunlight. An ointment containing disodium naphthal sulfonate is applied to screen out the ultraviolet rays in sunlight. Sulzberger and Wolf in one of their reports, recommend the following prophylactic remedy for sunburn:

Tan ic acid	20
Sulfol	20
Alcohol q.s. ad	1000
Sig. Apply before exposure to sunlight.	

Lotions are preferable to oils and ointments for treating sunburn because oils and ointments favor the development of pyoderma which is a sequela of sunburn.

The following bleach is recommended by Sutton:

Stearic chloride	1.0
Alcohol	25.0
Aqueous	74.0

Sutton and Sutton recommended the prophylactic use of a thick coating of zinc oxide or zinc powder liberally by talcum.

The face is cleaned of oil once or twice each day with benzene. The above-mentioned lotion is then applied three or four times each day. The provocation of an acute dermatitis is guarded against while employing this preparation.

Treatment. Vitamin A and cod liver oil are of value in diminishing the dryness of the skin. The larger and more noticeable lesions are destroyed by electrocoagulation. Care is taken to avoid scarring because a perfect cosmetic result is desirable. Every pigment cell is completely destroyed. Treatment by electrolysis and carbon dioxide is condemned, as is the use of peeling pastes or lotions.

Dermatitis Ambulationis

SYNONYM: Thermal burns.

Dermatitis ambulationis is a dermatitis of various degrees of intensity which is caused by excessive heat.

A scald is a burn that is produced by hot liquid or hot vapor; the effects of which are more extensive but less severe than burns which are occasioned by dry heat.

Varieties. The common classification of burns, depending on depth of skin damage, consists of three so-called "degrees." *First-degree burn* is also known as dermatitis ambulationis erythematosa, *second-degree burn* is known as dermatitis ambulationis bullousa, *third-degree burn* is referred to as dermatitis ambulationis escharacea.

Symptoms. Burns of the *first degree* are characterized by a transient erythema which is accompanied by local heat,

edema and pain and followed by epidermal desquamation.

In addition to the symptoms of a *first-degree burn*, *second degree burns* are accompanied by serous exudation with the formation of vesicles and bullae.

Burns of the *third degree* are characterized by symptoms of the first and second degree burn plus necrosis of both epithelial and connective tissue.

Constitutional involvement varies with the degree and extent of injury. Prostration, nervousness, vomiting and severe headache may be present in the milder cases. In extensive burns (anything above 15 per cent of the body surface) constitutional symptoms are present. There are varying degrees of shock during the first forty-eight hours. In burns which are severe, the nerve endings are usually damaged and pain is often absent, although shock supervenes in a few hours. In severe burns, the patient should therefore be treated for shock at once whether it is present or not. The onset of shock usually follows burns very soon after injury; it is rarely delayed for a few days. Shock is characterized by extreme prostration, low blood pressure, subnormal temperature, rapid, weak pulse and a moist cold skin. These symptoms may increase until death, which usually takes place during the first few days. In other cases, the symptoms of shock gradually disappear and the patient survives.

Complications. Complications comprise pyemia, erysipelas, tetanus, bronchopneumonia, pleurisy, nephritis, duodenal ulcer and septicemia.

Scars from burns are often keloidal. Scars which are the result of burns are more apt to have carcinomatous changes than any other type of scar.

Prognosis. A burn is usually fatal if it comprises one-third of the skin sur-

face, particularly if the chest or abdomen is involved

The prognosis depends on the promptness and effectiveness of treatment, as well as the extent of injury

Treatment In mild localized burns soothing lotions are all that is needed. Large blebs should be opened and drained. Dressings should not be changed more than once in twenty-four hours unless infection should be present. A 10 per cent solution of bicarbonate of soda or a half saturated solution of magnesium sulfate is beneficial, and being hypertonic their application minimizes the tendency to vesiculation.

The following lotion is a comfortable dressing for recent burns

Oil eucalypti	50.0
Oil olive	450.0
Liquor calci hydrox	500.0

Sulfadiazine Spray The treatment used at the Johns Hopkins Hospital is more effective than other treatments heretofore described and consists of spraying the burned areas with sulfadiazine. Sulfadiazine for the spray consists of an aqueous solution containing 3.5 per cent sulfadiazine and 8 per cent triethanolamine. Patients with severe burn are treated for shock and put to bed on sterilized sheets under a cradle heated with blue bulbs the temperature of the cradle should not exceed 90° F. The sulfadiazine spray is used every hour during the first day every two hours during the second day every three hours during the third day and every four hours during the fourth day. By this time a thin transparent crust forms, through which the physician can watch the healing of the burn and the growth of new skin tissues. The crust, though pliable, is strong and tough so that it does not break, and the patient is encouraged to exercise and to use his arms,

legs, or other burned parts of the body. This keeps the skin and tissues from contracting. After about ten days, the edges of the crust begin to loosen and separate from the skin beneath. At this time, sulfadiazine compresses may be applied and in many cases sterile mineral oil sprays, followed by isotonic salt solution compresses, allow the crust to be removed in large sheets. In third-degree burns the scab is left in place for at least two weeks.

Patients having burns of second-degree severity and not involving more than 20 per cent of the body surface are not hospitalized. Sulfadiazine spray is used frequently for two hours after injury and the burned area is dressed with either sterile petrolatum gauze or a sulfadiazine ointment.

Sulfadiazine ointment consists of 5 per cent sulfadiazine and 8 per cent triethanolamine in a stearin base (greaseless cream).

The sulfadiazine solution has a pH value of about 8.7. It is clear and has a faint yellow color that may darken unless stored in dark bottles. It does not stain, is almost odorless, has a bitter taste, does not injure skin, mucous membrane or granulating surfaces, and can be used safely in and around the eyes. It may be detected in the blood within several hours after having been sprayed on burned surfaces. For this reason, daily blood tests for sulfadiazine concentration in patients receiving multiple daily sprayings are advised. After the crust has formed the amount of the drug in the blood rapidly approaches a minimum even though spraying is continued, and the drug can then be given by mouth if desired.

Koch's Method. The burned surface, if infected, is cleansed with warm salt solution and white soap (this task re-

quires soft cotton) Nonadherent fine-mesh gauze, impregnated with simple petrolatum, is placed upon the lesion. This initial layer is then covered with gauze and mechanic's waste to a thick ness of from 4 to 6 inches. A muslin or Ace compression bandage is now applied, without restriction or constriction of the circulation. When possible, the part is supported by a splint. The compression bandage is not removed for from ten to fourteen days, unless foul odor or sudden rise in temperature and pulse rate suggests infection. Plastic repair is performed as early as feasible.

Management of Shock and Toxemia in Severe Burns: At present, plasma transfusions appear to be the best method of combating shock or fluid shift. It is the circulatory failure which begins within a few hours and persists for two or three days after the burn, it is caused by a leakage of plasma from the vascular compartment into the tissue spaces. Although some fluid is lost externally from the burned surface, most of the plasma lost from the blood stream is pooled in the tissues. This results in hemoconcentration and circulatory failure due to diminished plasma volume, as well as edema at the site of the burn.

Despite extreme hemoconcentration, the burned patient is not dehydrated, consequently giving large amounts of water by mouth or glucose solution intravenously aggravates edema without improving the circulation. *Intravenous saline* is beneficial in limited amounts only since excessive amounts of saline produce edema and further reduce the level of plasma protein. But when plasma is given after the capillaries recover there is a restoration of fluid balance. The plasma protein given in a plasma transfusion draws out fluid from the tissues and restores the plasma volume

to normal. The excess fluid is eliminated through the kidney.

Plasma is preferred to whole blood because the need is for plasma protein rather than for erythrocytes, and the requirement for plasma varies with each case. The less severe cases may respond satisfactorily to transfusion, totaling 500 cc. of plasma. In very severe cases, a continuous transfusion requiring 2 or 3 liters of plasma may be necessary. Lee *et al.* point out that, as a working rule, one may give enough plasma to keep the hematocrit value between 50 and 55 per cent cells and the plasma protein level about 8 gm. per 100 cc. until the capillaries return to a normal state of permeability. Then enough plasma is given to restore the plasma volume to normal.

Adrenal cortex extract (Upjohn) has been recommended for the treatment of burn shock. At the present time, say Lee *et al.*, "The mechanism of its action is unknown and its use is purely empiric." This hormone gives best results, it is felt, when used in conjunction with plasma transfusions. The dose for adults is 5 or 10 cc. of suprarenal cortical hormone given intravenously at six hour intervals for thirty-six or forty-two hours following the burn. Saline must be restricted to 1 liter during the cortical-hormone administration unless there is hypochloremia. Additional fluid is given in the form of glucose solution or plasma. *Penicillin* is of prophylactic value.

Dermatitis Autotoxica

Dermatitis autotoxica is a dermatitis resulting from metabolic disturbances, endocrine anomalies, pregnancy nephritis, gastrointestinal pathology, icterus, etc.

The variety of skin lesions depends on the organ that is affected. In hepatic disease, cutaneous xanthomas are not

uncommon. In diabetes necrobiosis lipodica may occur. In carcinoma of the adrenals acanthosis nigricans frequently occurs. In disease of the pituitary body acromegaly is a symptom. In disease of the thyroid follicularis keratosis may occur etc. These symptoms are described under the various titles which have been ascribed to them.

Dermatitis Congelationis

SYNONYMS: *Congelatio, frostbite*

Congelatio is the result of exposure to extreme cold. This condition results only after the tissues have been exposed to cold for a certain length of time. The objective symptoms are only visible after a period of latency. Berson and Angelucci state that the critical temperature for cooling tissues is in the region of 5 to 7° F. Tissues cooled below this temperature are killed.

Cold primarily damages the blood vessels producing an ischemia. The resulting symptoms are secondary to this primary damage and histological examination shows degenerative changes in the blood vessels. The vascular origin of congelatio in contrast to burns is evidenced by their unusually symmetrical appearance where the circulation is slowest, namely the hands, feet, nose and ears.

Becker and Obermayer divided congelatio into three degrees, namely (1) erythema, (2) stage of bullae formation and (3) necrosis and gangrene.

Symptoms. *First-degree* congelatio or freezing starts with a painful sensation of the affected part which is soon followed by loss of sensation. The first clinical sign is blanching of the skin which is caused by vascular constriction. If circulation is immediately restored by friction with snow the skin will again

become normal. If on the other hand the skin is exposed for a longer time, erythema and edema will result. The edema gradually subsides and the skin exfoliates and becomes normal with the possible exception of a degree of vascular paralysis which persists indefinitely.

Second-degree freezing develops in the same manner and is characterized by a livid erythema and the formation of bullae. The bullae are not as sharply outlined as those resulting from a burn and do not have the same clear content. Their color is often dark due to the admixture of blood. In mild cases, the bullae rupture, leaving superficial excoriations followed by crusting. They heal in a few days without scars.

Third-degree freezing is characterized by the presence of necrosis and gangrene. Bullae may not necessarily precede these characteristics. The extent of the permanent damage depends on how much of the vascular supply has been destroyed. The dead tissue finally separates and the involved portion occurs either as dry or moist gangrene. In extensive cases, the entire extremities may be lost.

Etiology. Congelatio of the first and second degree occurs most frequently in regions where the blood vessels are most exposed, such as the nose, ears, cheeks, hands, and feet.

Third-degree freezing occurs most frequently on the feet. Lowered vitality of the extremities, varicose veins, ill fitting shoes, and infrequent changes of shoes and socks are contributory etiological factors.

Prophylaxis. Warm clothing, woolen stockings, gloves and mittens, and shoes that are not too tight are prophylactic measures.

Prognosis. The prognosis as to life is generally good.

Treatment First-degree freezing responds favorably to gentle *massages* with snow or cold water. The temperature of the affected parts should be raised gradually.

Second and third degree freezing should be treated with *gentle massage*. Constricting shoes should be removed and walking prohibited. Use no local warming agents. Irritating applications should be avoided. *Aseptic dressings* are advised. Internally *warm drinks* and moderate *stimulation* are helpful adjuncts. The treatment must always be conservative; the goal of treatment is the avoidance of gangrene and secondary infection.

Chilblain

SYNONYMS *Pernio*.

Exposure to cold causes injuries in various degrees, the most common being chilblain, or *pernio*.

In those predisposed by poor peripheral circulation even moderate exposure to cold may produce chilblain.

Chilblains are characterized by a localized erythema and edema. The sites of predilection are the hands, feet, and ears. The subjective symptoms are burning, itching, and tenderness. The affected areas are bluish-red and cool to the touch. The color of these areas disappears on pressure. A feeling of clamminess due to excessive sweating may be present. The disorder is seasonal, being present in winter and absent in the summer. If exposure to cold is prolonged, nodules and plaques develop on the surface and hemorrhagic bullae may form. Central necrosis occasionally follows. *Perniones* are frequently seen in England and on the Continent and rather infrequently in the United States. Natives of India sojourning in England do not develop *perniones*, which indicates that

exposure to cold is not the sole causation.

Acrocyanosis or *acrosphymia*, is a functional circulatory disturbance resulting in cold, usually perspiring hands and feet, associated with varying degrees of color change. The skin is reddish blue often puffy always cold and usually moist. The color change is often sharply demarcated above the wrist or ankle. It is commoner in females. It often develops at or shortly after puberty. It may be associated with effort syndrome, hypothyroidism, ovarian dysfunction, or debilitating diseases. Lewis considers it a capillary anoxemia due to angospasm. The term *erythrocyanosis* has been used for a somewhat similar condition observed on the lower extremities of hemiplegics, the palsies of infantile paralysis, and in girls and young women in conditions not well understood. The affected parts are cold, bluish, and have an edematous-like fullness but there is no pitting on pressure. Indurated nodules may form in the skin and break down or they appear to simulate *erythema induratum*. These latter lesions may be due to localized attacks of thrombophlebitis.

Prophylaxis The wearing of woolen clothing and gloves is a prophylactic measure.

Prognosis The prognosis is guardedly favorable. Removing the patient to a mild climate hastens recovery.

Treatment The treatment is general as well as local. A high caloric as well as a high vitamin diet is essential. Generalized ultraviolet light therapy is beneficial. Such drugs as arsenic and *calcium gluconate* are occasionally helpful. The

affected parts should be bathed in water of varying degrees of temperature and gently massaged with warm oil. Painting the affected parts with a solution of *ichthol* and *tragacanth* (*ichthol varnish*) is usually well tolerated.

Dermatitis Exfoliativa

SYNONYMS: *Pityriasis rubra, exfoliativa dermatitis erythroderma exfoliativa (Besnier) dermatitis exfoliativa (Besnier)*

Dermatitis exfoliativa is an acute or chronic extensive cutaneous inflammation characterized by redness, desquamation, and itching.

Incidence: Dermatitis exfoliativa is most frequent in middle life and males

Toxemias, foci of infection, leukemia, Hodgkin's disease, tuberculosis, rheumatic states, and other debilitating diseases are predisposing causes. Dermatitis exfoliativa may also accompany contagious diseases. A toxic erythema resembling dermatitis exfoliativa occasionally appears between the seventh and fifteenth day of an attack of smallpox.

Pathology: The histopathology of dermatitis exfoliativa consists of marked

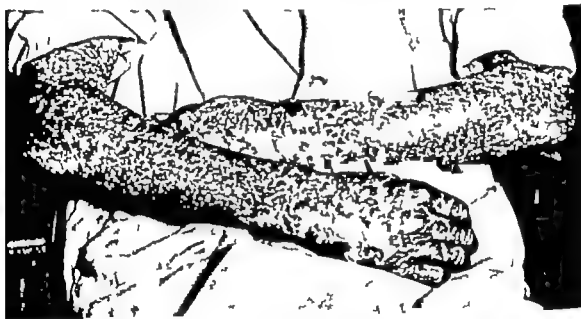


Fig. 179: Dermatitis Exfoliativa. Following arsenphenamine therapy

are more often affected than females in the ratio of three to one.

Etiology: Dermatitis exfoliativa is often a transition from some chronic dermatosis like psoriasis, lichen planus, and seborrheic dermatitis. The condition arises without any demonstrable cause in connection with overenergetic local treatment of the skin. The application of certain chemicals like chrysarobin, mercury, and tincture of iodine to the skin as well as the intake of such drugs as quinine, arsenic, antipyrine, the arsenphenamines, gold bismuth insulin serums, etc., produce desquamating erythrodermia in hypersensitive individuals.

vascular dilatation about the hair follicles and sweat glands. Interstitial and parenchymatous edema of the upper part of the corium and epidermis is present. The papillae are thickened and elongated. Acanthosis and parakeratosis are present in the interpapillary segments. The granular layer is absent. Marked atrophy of the papillae and sebaceous glands occurs in the more chronic cases.

Symptoms: Two types of dermatitis exfoliativa are recognized. A primary type of dermatitis exfoliativa is of sudden onset and accompanied by malaise and fever. One or more hyperemic patches of dermatitis appear spread rap-

kly and may be followed by a generalised distribution. The reddened skin begins to scale within a few days. Flaking is rather large and abundant. The soles and palms usually peel off in casts. The skin is dry and feels tender and tight. Itching is slight. The patient complains of chills throughout an attack. The hair is shed and the nails may undergo exfoliation and deformation as the disease becomes chronic. The mucous membranes may become involved. The disease may persist for weeks, months, or years with recurrences. Some cases of short duration have been designated as erythema scarlatiniforme and the recurrent types as recurrent desquamative scarlatiniform erythema.

The secondary type of dermatitis exfoliativa develops gradually. This variety often follows eczema, psoriasis, and seborrheic dermatitis, or it may follow the use of applications containing mercury, chrysarobin, arsenic, etc. It is not distinguishable from the primary type when fully developed. There may be considerable swelling about the lips, nose, eyelids, and genitalia in severe cases. Oozing, paroxysmal attacks of itching and fever are often present. Hyperpigmentation is a marked feature. Glandular enlargement may also be present.

Pityriasis Rubra (Hebra) This is a chronic and rare variety of dermatitis exfoliativa. The entire cutaneous surface is involved. The scales are small, infiltration is absent and a glossy atrophy ultimately supervenes. The face and extremities and finally the entire body is the seat of a vivid scarlet erythema which is covered with small laminated scales which exfoliate freely. Vesiculation, pustulation, oozing, and itching are absent. Constant chilliness usually accompanies the disease. Death results from exhaustion or bronchopneumonia.

Epidemic Exfoliativa Dermatitis (Savill's Disease, Epidemic Eczema) In 1891 Savill described an eczematoid affection which had occurred as an epidemic in London workshops during the autumn months. Similar outbreaks have been reported by Fordyce and Winfield in this country.



Fig. 180 Dermatitis Exfoliativa. Following streptococcal therapy.

Savill described two varieties: the first was moist and resembled eczema; the second was dry and resembled pityriasis rubra. The cases occur in middle life or later. The eruption consists of purplish or reddish macules, or papules which coalesce to form patches which extend peripherally until the entire cutaneous surface is involved. Exfoliation commenced early and continued for four or five weeks. The course of the disease is about eight weeks but recurrences are frequent. Death results in the aged and very debilitated.

Diagnosis The history mode of on-

set and course of the disease differentiate it from other diseases which may simulate it. The primary patch of psoriasis is infiltrated and has silvery mica like imbricated scales beneath which are minute red points consisting of capillary loops. The typical lesion of lichen planus is raised flat and has a striated violaceous surface with angular border. The patch of seborrheic dermatitis is only superficial yellowish in color and with out abundant scales. Scaling in chronic eczema is scant. Dermatitis scarlatinoides has a sudden onset and is of a shorter duration. The throat is red and sore in scarlet fever.

Prognosis. The prognosis is favorable. Recovery occurs in the majority of patients who are not debilitated. Aged patients usually manifest grave constitutional symptoms and usually die.

Treatment. The causative factor is treated when possible (see therapy for arsenical intoxication p 283). The administration of vitamin B complex over a long period of time is the best known internal medication. Walker lauds small and frequent doses of wine of antimony.

Quinine, thyroid extract and ephedrine are of value.

The patient is kept in bed in a warm room during the acute stage and precautions must be against undue exposure to wind and weather. The diet must be rich in vitamin content. Alcohol is strongly contraindicated.

Local treatment consists of applications of bland ointments and lotions such as petrolatum, boric acid, White's crude coal tar, lanolin and carron oil with 5 per cent ichthyol. Tepid bran and starch baths are beneficial. Fifty per cent cod liver oil in paraffin and petrolatum is occasionally very helpful.

Andrews advises irradiation by fractional doses of unfiltered x rays.

Dermatitis Exfoliativa Neonatorum

SYNONYMS: *Ritter's disease*
keratolysis neonatorum, *dermatitis exfoliativa infantum*.

Dermatitis exfoliativa neonatorum is a staphylococcus infection of the skin of nurslings.

Incidence. The disease is rare and usually occurs between the second and eighth week of life. It is usually seen in foundling asylums.

Etiology. The cause of this disease is still obscure. It is, however, regarded as a dermatitis of pyrogenic origin. It is quite probable that a primary vitamin deficiency precedes the infection which is superimposed on this abnormal and nonresistant skin.

Pathology. Extensive areas of the skin are edematous and reddened, and the epidermis is sloughed off in masses. Linear hemorrhages occurring between the epithelium and papillae account for the blue striae seen clinically in this disease.

The corium shows marked edema of the papillary bodies and the subpapillary layer and the dilated vessels are filled with red blood cells. The dissolution and exfoliation of the upper portions of the epithelium are the most characteristic changes.

Symptoms. The disease is rare and usually begins as vesicles which spread peripherally and become confluent, thus involving large areas. When the vesicles rupture, intensely reddened areas result. It usually starts on the face and mouth, more especially on the chin. It spreads from the face to the neck and trunk. The extremities are rarely involved. The erythema is of rapid progress and is accompanied by dryness and exfoliation. The loosened horny layer of the skin

wrinkles and is readily removed by pressure of finger and clothes. Vesicles and bullae identical to those of periphagus neonatorum may appear early. Some clinicians are of the opinion that the lesions of dermatitis exfoliativa neonatorum are not distinguishable from those of periphagus neonatorum. The disease may exhibit flaking and crusting of the mucous membrane of the mouth. The conjunctivae are usually congested. Elevation of temperature and other symptoms of systemic involvement are more or less severe and occur in proportion to the amount of body involvement.

Diagnosis Diagnosis should cause no difficulty. It must be differentiated from erythroderma exfoliativa (Leiner's), pityriasis rubra, erythroderma ichthyiforme congenitum, epidermolysis bullosa congenitalis, and congenital lues.

Leiner's disease usually begins as a seborrheic dermatitis and it usually disappears after three or four weeks. The eyelids in this disease are greatly swollen and covered with scales. Bullae and eczemas are not present.

Complications These include stomatitis, rhinitis, corneal ulcers, subcutaneous abscesses, gangrenous lesions, and bronchopneumonia.

Prognosis The prognosis is unfavorable. Mild cases usually clear up in a few weeks. The mortality is about 80 per cent.

Prophylaxis A diet well fortified with vitamins A and B complex is a prophylactic measure.

Treatment The treatment consists of the oral administration of vitamin B complex with riboflavin. In addition to this, injections of aut serum or taphylococcal vaccine are indicated. Locally an ointment containing 80 per cent cod liver oil, U.S.P. in a base of petrolatum

and paraffin is applied to the skin. The eroded corners of the mouth should be treated with 0.25 to 0.5 per cent solution of silver nitrate. Bathing is contraindicated at the beginning, although potassium permanganate 1:8000 or tannin 200 gm to a full bath, is of service after the child's skin shows less exfoliation.

Very painful lesions may require soothing ointments, such as

Pet. linc. adjuvante	20.0
Pur. zinc oxide	40.0
Oil of	40.0

A 3 per cent solution of boric acid is useful in the treatment of the inflamed conjunctivae. The body heat must be maintained by light covers or an electric pad. The patient should be completely isolated.

Dermatitis Facilita

SYNOCTYMS *Dermatitis artefacta, malingering, feigned or self-inflicted injuries, hysterical gangrene, neurotic gangrene.*

Dermatitis facitila identifies self-inflicted cutaneous lesions acquired with the intent of attracting sympathy, escaping duty or collecting insurance.

Etiology The subjects of dermatitis facitila are neurotic and hysterical young women, although men are by no means free from this condition. The lesions are provoked by mechanical means, bites, fingernails, sharp instruments, glass, hot metal, or by the application of irritating chemicals such as phenol. Gillespie differentiates the true hysterical states from the more commonplace examples of malingering, attention-getting desire for sympathy obtaining certain privileges, and similar motivations. Hysterics may not know why they produce the lesions, but many are aware that they actually do so.

Occupational neuroses of the skin are based on the patient's desire for additional consideration and the patients prolong the illness by exaggerating the objective and subjective signs.

Symptoms Inflicted lesions may simulate other dermatoses but possess a distinctive, clean cut, bizarre appearance. The shape and arrangement of

self produced lesions are of a nature not encountered in any other skin disease. The lesions are linear arranged regularly and of symmetrical distribution over parts of the body easily accessible to the hands. They are rarely seen on the right hand and right wrist, or even right side of the body unless the patient is left handed. The eyelids, scalp, interscapular region and soles are not mutilated. Loss of the pharyngeal reflex is common in patients with feigned myoclonus. It is probable that the so-called "hysterical pemphigus" (bullous lesions) and "hysterical gangrene" are examples of dermatitis factitia.

Diagnosis If the diagnosis is in doubt, it can occasionally be proved by applying litmus paper to a recent lesion to detect the presence of acid or alkali. The patient should be prevented access to the affected parts by an occlusive dressing. The best occlusive dressing is one made with hard zinc oxide paste (N.F). Prompt healing would naturally incline one to the opinion of dermatitis self inflicted.

Treatment It is sometimes difficult to be sure of the diagnosis. In addition, it is often difficult to convince the families



Fig. 181: Dermatitis Factitia. At site of scar on lower abdomen.



Fig. 182: Dermatitis Factitia. (Courtesy of Dr. J. V. Klunder)

of these patients that the lesions are self-inflicted. If a definite psychoneurosis is present, psychiatric treatment should be sought.

The local treatment of the lesion depends on the type of lesion present.

Dermatitis Gangrenosa

SYNONYMS *Sphaceloderma*,
cicatricial gangrene.

Necrosis, or death of a cutaneous part of the body of varying extent produces a characteristic clinical appearance termed gangrene. Clinically it may be the only objective condition present, or it may be part of an ulcerative process. Ulceration is present, however, in all cases in which the necrotic area shows signs of detachment.

Etiology and Classification. Cutaneous gangrene may develop in the course of a variety of diseases. For practical purposes, Brocq suggests the following etiological classification:

1. Traumatic
 - (a) Primary gangrene
 - (b) Secondary gangrene

TRAUMATIC GANGRENE. This may be due to:

1. **Mechanical Agents.** Such as prolonged pressure or actual direct injury of a contusing, crushing, or tearing type.

2. **Physical Agents.** Heat (burns) cold (frostbit) excessive radium or ray exposure and electric currents from a variety of sources.

3. **Chemical Agents:** Strong acids and alkalis, phenol dressings to dependent parts, arsenical pastes, bichloride of mercury iodides, and zinc chloride.

NOTTRAUMATIC GANGRENE: Primary Gangrene. This is seen

1. Following ingestion of certain toxic substances, such as ergot (ergotism) chloral hydrate, and carbon monoxide

In chronic ergotism, the gangrene affects the extremities, usually the toes and fingers, sometimes the ears and nose.

2. As a result of (a) vascular lesions interfering with blood flow such as are seen in thrombosis, embolism, decubitus, arteriosclerosis (senile gangrene) thromboangiitis obliterans (see p. 739) and



FIG. 188. Gangrene following exposure to extreme cold.

gangrene of the extremities (Raynaud's disease; see p. 657) (b) in hysterical gangrene; and (c) pressure of a newgrowth or cicatricial structure. The exact mechanism of gangrene as it occasionally occurs in malignant growths and the tumors of myxomatous fungoides is not clear.

3. In certain general diseases, such as diabetes mellitus.

4. In conditions caused by bacteria, individually or in symbiosis, usually in skins whose resistance has been lowered by local injury or systemic diseases, such as malaria, typhoid, pneumonia, scarlet fever or chronic renal, cardiac, pulmo-

nary or hepatic disease. These forms of gangrene include ecthyma gangraenosum, multiple infectious gangrene of adults, and fulminating gangrene of the genitalia, as well as cancrum oris (noma).

GANGRENE IN DIABETES. Gangrene in diabetes may be primary or secondary

black and may follow either a rapid or a slow course. Frequently diabetes is not known to exist until after the development of gangrene.

Prognosis. If the gangrene is limited to a small area the prognosis is good. In extensive cases and especially in the presence of sepsis, as manifested by temperature elevation, general malaise, chills, and sweats, it is serious. The insulin requirements and the level of sugar in the blood are important guides to the amount of toxicity resulting from a local lesion. The prognosis in gangrene of the toes is improved by a good dorsalis-pedis pulse and the absence of pain.



Fig. 184. Gangrene. Result of radium dermatitis.

to an injury: furuncle, carbuncle, or abscess. It is not uncommon and may be frequently observed in patients with diabetes who are in apparently good health and who do not have an abnormally large amount of sugar in the blood. In the primary types it is generally believed to develop as a result of peripheral vascular inflammation and decreased blood supply rather than as the result of hyperglycemia. Infection plays a secondary part in the moist types. The lesions usually appear on toes and fingers, or as necrotic lesions on the legs and feet, often spontaneously or following a slight injury. They are frequently in the form of bleb-covered areas, ranging from grayish brown to



Fig. 185. Diabetic Gangrene.

Treatment. Control of the diabetes may be of value, but it is not always easy to obtain. In certain cases, early amputation may save life. Except for very localized areas, considered judgment is necessary in both the dry type and the moist type especially to deter

mine whether conservative or radical procedures should be chosen. Amputation is almost certainly indicated if the necrosis has involved the deeper tissues. In all cases, it is important to maintain the patient's water balance and nutrition.

ECTHYMA GANGRAENOSUM This is discussed elsewhere (see p. 310)

MULTIPLE INFECTIOUS GANGRENE OF ADULTS The lesions appear primarily on a healthy skin and secondarily on a preexisting lesion (impetigo, syphilides). It is rare but appears to be similar to that seen in infants and described under *Ecthyma Gangraenorum* (p. 310). The onset, symptoms, and course are practically the same. The lesions in some are contagious and autoinoculable. A variety of organisms, especially streptococci, have been isolated, both aerobic and anaerobic. The patient's general condition may become serious.

FULMIGATING GANGRENE OF THE GENITALIA (Pournier) This is exceedingly rare and occurs commonly in the young adult. The onset is sudden, with chills, fever and malaise in a patient whose sole lesion is an erythematous spot on the penis. Redness increases, and the affected part rapidly becomes edematous. The penis may swell to a enormous size. Vesiclobula, containing reddish serum, appear on the shaft of the penis in from twenty-four to forty-eight hours. Sometimes the erythematous spot becomes placoid and anesthetic and may be so from the outset. The scrotum and draining lymph nodes are often involved. Delirium and coma intervene and the general condition becomes grave. In favorable instances the general disturbances disappear and the necrotic area heal and cicatrize in the usual manner but with considerable mutilation of the parts.

Secondary Gangrene This develops

in the course of certain infectious processes like herpes zoster chancreoid, erysipelas, leprosy, tuberculosis, and syphilis. Included here are those types of gangrene occurring in the course of marked cachexia and serious disease of the nervous system, such as syringomyelia or Morvan's disease. In many of these, septic emboli—containing anaerobic organisms derived from such sources as bed ulcers and pulmonary gangrene—appear at times to be the factor.

Symptoms of Gangrene in General
Early Symptoms When gangrene is about to develop, the first changes are in the local skin temperature, sensation, and color. In the moist type, the skin is grayish and, on the affected places, one or several blebs appear filled with a reddish serum. In the dry type the skin ranges from yellowish brown to black, the part appears to retract and sink somewhat below the normal skin level, and the affected area is firm and anesthetic. In some, there are initial and persistent subjective symptoms—tingling, burning, and even severe pain.

Later Symptoms. In several days or longer the tissue surrounding the gangrenous area develops a more or less red inflammatory zone. A groove separating the dead from the living tissue forms at this point. The necrotic area slowly contracts and separates, exposing an ulceration in which granulations appear. Complete, progressing separations follow progressive healing and ultimately cicatrization.

Treatment of Gangrene in General
Generally This depends on the part affected as well as on the predisposing and actual factors. Each case must be individualized, however without too much dependence being placed on systemic therapy. Locally the affected part is kept as clean as possible. Blebs are

opened and drained, and the areas washed with mild *antiseptic solutions* such as boric acid phemerol (1 1000) mercurophen or metaphen (1 1000). Moist wet dressings with these solutions favor separation of the necrotic area. After these procedures—and in moist types—*powders* (thymol iodide sulfa thiazole, activated zinc peroxide) are preferable and favor cicatrization. In extensive septic involvements, destruction just beyond most of the invasive area by means of *electrocoagulation* has been successful. In definitely infectious types *penicillin* both locally (as wet compresses, 500 units to each cubic centimeter of sterile distilled water) and intramuscularly (30 000 units every three hours to a total dosage of 1 000 000 units) has been used with benefit. *Amputation* may be necessary.

Dermatitis Hemostatica

SYNONYMS: *Dermatitis hypostatica*

Dermatitis hemostatica is characterized by varicolored pigmentary changes of the skin of the ankles, legs, or arms. Slight edema may accompany this phenomenon.

Etiology Passive congestion is the causative factor.

Pathology Venous dilatation is the most significant histological change that is found.

Symptoms The objective symptoms consist of a symmetric circumscribed livid redness located on the extensor surfaces of the legs and arms. No subjective symptoms are present (see varicose veins, p 815).

Prognosis A retrogressive atrophy may occur and the condition may at least partially disappear.

Treatment No specific treatment has been reported. *Roentgen rays* may be beneficial.

Dermatitis Herpetiformis

SYNONYMS: 'Duhring's disease, *Impetigo herpetiformis*, *herpes gestationis*, *dermatitis multiformis*, *hydra herpetiformis*, *penphigus prurigenosus*.

Dermatitis herpetiformis is a chronic pruritic disease of the skin characterized by successive outbreaks of polymorphic lesions with a preponderance of small vesicles which are grouped in clusters.

Varieties Herpes gestationis is a variety of dermatitis herpetiformis occurring in pregnant women. Hydra puerorum (Unna) is a variety seen in young children which disappears, recurs (especially during the summer) and finally fades completely around puberty. In the vesicular form of dermatitis herpetiformis, the majority of the lesions are vesicles or small bullae. In the nonvesicular variety the predominant lesions may be macules, papules, pustules, erythematous plaques, or urticarial wheals.

Incidence The majority of patients suffering with dermatitis herpetiformis are high strung nervous, poorly nourished and debilitated. It is met with in both sexes and more often during adult life, although it occasionally occurs in children.

Etiology The cause of this disease is unknown. Some cases of dermatitis herpetiformis are undoubtedly allergic manifestations. Such allergens include bromides, iodides, salicylates, chocolate, fish streptococci etc. A dysfunction of the thyroid gland may be the causative factor. The favorable effects of the sulfonamides on the eruption suggest a hypersensitivity to bacteria or their products. These patients also show a cutaneous hyperactivity to many types of bacterial extracts (staphylococcus, streptococcus, tuberculin, trichophyton, and colon bacillus).

Pathology The polymorphism of the eruption in this disease makes it impossible to provide a definite histological picture. Clinically the eruption may include any one or all of the following lesions: erythema, papules, vesicles, pus-

ment function of the basal cells and of the chromatophores in the corium.

Symptoms Frequently the first symptom of the disease is the eruption, but it may be ushered in by mild constitutional symptoms which are well



Fig. 186 Hydrum Patersoni (Dahring disease) In boy

ules, wheals, or nodules. The histology therefore depends upon the type of the lesion examined. The inflammation is limited to the papillary layer while the deeper dermis shows little change. In the dermis, eosinophils are found in the blood vessels and lymph spaces. Healing lesions show a great increase in the pig-

mented in severe cases. The eruption is usually symmetrically distributed and the lesions appear suddenly in groups. The skin over the scapular regions, the sacrum, the lateral surface of the thigh, and the extensor surfaces of the upper arms and forearms are sites of predilection. The lesions of one attack may be

opened and drained and the areas washed with mild *antiseptic solutions* such as boric acid phemerol (1 1000) mercuraphen or metaphen (1 1000). Moist wet dressings with these solutions favor separation of the necrotic area. After these procedures—and in moist types—*poultices* (thymol iodide, sulfa thiazole activated zinc peroxide) are preferable and favor cicatrization. In extensive septic involvements, destruction just beyond most of the invasive area by means of *electrocoagulation* has been successful. In definitely infectious types *penicillin* both locally (as wet compresses 500 units to each cubic centimeter of sterile distilled water) and intramuscularly (30 000 units every three hours to a total dosage of 1 000 000 units) has been used with benefit. *Amputation* may be necessary.

Dermatitis Hemostatica

SYNONYMS: *Dermatitis hypostatica*.

Dermatitis hemostatica is characterized by varicolored pigmentary changes of the skin of the ankles, legs or arms. Slight edema may accompany this phenomenon.

Etiology Passive congestion is the causative factor.

Pathology Venous dilatation is the most significant histological change that is found.

Symptoms The objective symptoms consist of a symmetric circumscribed livid redness located on the extensor surfaces of the legs and arms. No subjective symptoms are present (see varicose veins, p 815).

Prognosis A retrogressive atrophy may occur and the condition may at least partially disappear.

Treatment No specific treatment has been reported. Roentgen rays may be beneficial.

Dermatitis Herpetiformis

SYNONYMS: *Dukering's disease*, *Impetigo herpetiformis*, *herpes gestationis*, *dermatitis multiformis*, *hydra herpetiformis*, *petaphigus pruriginosus*.

Dermatitis herpetiformis is a chronic pruritic disease of the skin characterized by successive outbreaks of polymorphic lesions with a preponderance of small vesicles which are grouped in clusters.

Varieties Herpes gestationis is a variety of dermatitis herpetiformis occurring in pregnant women. Hydra puerorum (Unna) is a variety seen in young children which disappears, recurs (especially during the summer) and finally fades completely around puberty. In the vesicular form of dermatitis herpetiformis, the majority of the lesions are vesicles or small bullae. In the nonvesicular variety the predominant lesions may be macules, papules, pustules, erythematous plaques, or urticarial wheals.

Incidence The majority of patients suffering with dermatitis herpetiformis are high strung nervous, poorly nourished and debilitated. It is met with in both sexes and more often during adult life although it occasionally occurs in children.

Etiology The cause of this disease is unknown. Some cases of dermatitis herpetiformis are undoubtedly allergic manifestations. Such allergens include bromides, iodides, salicylates, chocolate, fish streptococci, etc. A dysfunction of the thyroid gland may be the causative factor. The favorable effects of the sulfonamides on the eruption suggest a hypersensitivity to bacteria or their products. These patients also show a simultaneous hyperactivity to many types of bacterial extracts (staphylococcus, streptococcus, tuberculin, trichophyton, and colon bacillus).

Pathology The polymorphism of the eruption in this disease makes it impossible to provide a definite histological picture. Clinically the eruption may include any one or all of the following lesions, erythema, papules, vesicles, pus-

ment function of the basal cells and of the chromatophores in the corium.

Symptoms Frequently the first symptom of the disease is the eruption, but it may be ushered in by mild constitutional symptoms which are well



Fig. 186 Hydrum Paucorum (Dühring's disease) 1 boy

tules, wheals, or nodules. The histology therefore, depends upon the type of the lesion examined. The inflammation is limited to the papillary layer while the deeper dermis shows little change. In the dermis, eosinophils are found in the blood vessels and lymph spaces. Healing lesions show a great increase in the pig-

mented in severe cases. The eruption is usually symmetrically distributed and the lesions appear suddenly in groups. The skin over the scapular regions, the sacrum, the lateral surface of the thigh, and the extensor surfaces of the upper arms and forearms are sites of predilection. The lesions of one attack may be

of one type or at least one type may predominate and in the next recurrence they may present another type. The extent of the eruption varies from a few plaques to a generalized distribution. Burning or itching is almost invariably present and constitutes the patient's chief complaint.

The vesicular type of eruption is the most characteristic form; the grouped

by the constant development of new lesions, but as a rule the attacks occur in successive crops with periods of quiescence. Sometimes a few recurrent vesicles may be the only sign of the disease for a long time. There are cases on record in which lesions become vegetative so that some observers speak of a dermatitis herpetiformis vegetans. Nikolsky's sign may be present



Fig. 187. Dermatitis herpetiformis. Right: Of fifteen years duration. Note grouping of ectopapular lesions.

plaques of vesicles suggesting herpes simplex, hence the name; but the mixed type is the most common. In the vesicular, bullous, and pustular form the lesions have thick walls and seldom if ever rupture spontaneously but are destroyed by the patient's nails in order to obtain relief from the intolerable burning and itching. In children vesicular or bullous lesions predominate and a change in type is less frequent than in adults. An attack may be prolonged

In 20 per cent of cases, vesicular or bullous lesions may be present on the mucous membrane of the mouth, nose, esophagus, or genitalia. A variable degree of pigmentation commonly remains after the lesions have healed. As a rule, the general condition of the patient remains excellent in spite of the suffering. There are however rare cases on record which eventually terminated fatally through general debility and progressive loss of weight. Ordinarily the

disease lasts for a number of years and eventually disappears.

The administration of iodine has a provocative effect on subjects with pemphigus and dermatitis herpetiformis, especially the latter but the internal administration of iodine should not be used for diagnostic purposes. The same effect is reached if iodine is applied to the skin in the form of a patch test with an ointment containing 50 per cent potassium

show the same findings as in pemphigus; however in dermatitis herpetiformis, eosinophilia is common. The retention of sodium chloride is present in dermatitis herpetiformis, though to a lesser degree than in pemphigus.

Herpes Gestationis. This rare condition does not differ clinically from Duhring's disease. It is characterized by vesicles and intense itching. The vesicles appear on an inflammatory base. The onset



Fig. 155 Dermatitis Herpetiformis. Right Note the characteristic grouping of the lesions.

iodide in petrolatum or a 20 per cent solution of potassium iodide. The test is often positive in the form of vesiculation even at times when no cutaneous eruption is present especially over areas where lesions previously had been present. This hypersensitivity to iodine may be present in pemphigus, in exfoliative dermatoses, and in some toxic eruptions; moreover even in dermatitis herpetiformis, the test is not always positive as positive and negative phases seem to alternate in the same patient.

The examination of the blood and of the content of the bullous lesions may

is usually during the second half of pregnancy more rarely at an earlier date. In some women it develops only during the puerperium. The abdomen is the site of predilection, and the lesions often begin around the umbilicus. Urticarial lesions may be part of the morphologic picture. There are patients who suffer from herpes gestationis with each pregnancy and others who develop it at later pregnancies. The essential point of differentiation is the fact that herpes gestationis is only present during pregnancy or the puerperium, while during the intervals the patient remains entirely free

from the disease thus suggesting a toxic or neurogenic origin. Cases are recorded of patients, who after suffering attacks of herpes gestationis during several pregnancies continued to develop the eruption after delivery as typical dermatitis herpetiformis. Eosinophilia is usually present just as in dermatitis herpetiformis. Testing of the skin however with iodides or bromides gives negative results. In severe cases termination of the pregnancy may have to be considered.

Diagnosis. Dermatitis herpetiformis must be differentiated from pemphigus vulgaris and the nonvesicular form from other itching dermatoses. The diagnostic symptoms and signs of dermatitis herpetiformis consist of burning and itching grouped arrangement of the lesions erythema polymorphism and symmetric distribution occurring in patients who are in fairly good health.

In pemphigus vulgaris, the eruption consists of bullae which are usually large, thin walled and exhibit no tendency towards symmetry and spring from a noninflammatory skin. Pruritus is usually slight or absent. In dermatitis herpetiformis, the bullae vary in size and may sometimes be large but usually they are small and arise on an erythematous plaque or are surrounded by an erythematous halo. In severe cases of pemphigus, the patient is seriously ill. In severe cases of dermatitis herpetiformis, the patient is not ill.

The nonvesicular forms of dermatitis herpetiformis must be differentiated from scabies pediculosis vestimentorum chronic urticaria erythema multiforme, and drug eruptions. The symmetry of the eruption the grouping of the lesions and the intense pruritus will suggest dermatitis herpetiformis and one should

watch for the appearance of vesicles which sooner or later are bound to be present.

Herpes gestationis occurs during pregnancy and puerperium.

Prognosis. The prognosis is guarded and complete recovery is rare. The outlook is favorable in children. Spontaneous recovery however does occur. Dermatitis herpetiformis usually continues for years without endangering health. Subjective symptoms can always be relieved.

Treatment. The administration of arsenic to the limit of tolerance is the treatment of choice. Arsenic is given as Fowler's solution or as arsenous acid. *Tryparsamide* and *neoarsphenamine* have been used with equal benefit. *Sulfapyridine* in 1-gm doses, three to four times daily is of great value. The dose is later reduced to a maintenance one only. Relapses occur when it is discontinued.

Two intramuscular injections of 10 cc. of the patient's own blood have been given each week with benefit. In herpes gestationis, subcutaneous injections of the serum of healthy pregnant women have been helpful. Injections of normal serum have also been advised. Quinine in the largest tolerated doses has been of value.

Injections of liver extract have also been beneficial. Roentgen rays to chronic localized areas are beneficial.

Physiotherapy and wet dressings of hot boric acid are definitely helpful. The colloid bath containing oatmeal, starch or bran is soothing. Sulzberger prefers baths of potassium permanganate, tar or sulfur. Patients on a milk and vegetable diet have derived benefits and remain free while on this diet from lesions for a long period.

Table salt, iodides, bromides, salicylates, chocolate, fish, shellfish, pork, and nuts are ruled out from the diet.

Gradual tanning of the skin by exposure to ultraviolet light, three times each week, diminishes itching and lessens the number of lesions.

In mild cases, dust the lesions at bedtime with antipruritic powders such as:

Menthol	20
Bismuth subgallate	100
Ammonium	100
Salicylic acid	1000
Sig: Dusting powder	

Duhring recommended an ointment containing 5 per cent sulfur precipitate and 1 per cent menthol.

An antipruritic lotion is the following:

Phenol	20
Menthol	10
Thymol	0.5
Alcohol 70 per cent q. s.	1000
Sig: Use for relief of itching	

Sodium thiosulfate intravenously administered in 1-gm doses daily for one week is sometimes helpful.

A complete change of environment often effects a cure.

Dermatitis Hiemalis

Dermatitis hiemalis is a dermatitis resulting from exposure to extreme cold, more especially to cold winds. The face and exposed parts of the body are involved. The lesions are erythematous and if the exposure to cold continues, symptoms of dermatitis congelationis (chilblains) supervene.

Treatment. Applications of ice water should be applied immediately after exposure. This should be gradually replaced by compresses of water of higher temperature until the normal temperature of the body is attained. Soothing lotions such as calamine liniment, may then be applied.

Dermatitis Infectiosa

SYNONYMS *Infectious eczematoid dermatitis*, Engman disease.

Dermatitis infectiosa eczematoides is an inflammatory disease of the skin characterized by vesicular pustular and multiform lesions occurring on circumscribed reddened patches of moderate size.

Incidence: The disease occurs more frequently in adults and in males more often than in females.

Etiology. The immediate cause is infection of the skin by one of several pyogenic organisms, more often the *Staphylococcus aureus*. It is usually secondary to a discharging abscess, sinus, or ulcer. It is often associated with chronic furunculosis, paronychia, middle ear disease, bedsore, fistula, or discharges from eye, nose, rectum, and vagina, or from mycosis of the toes. The affection spreads by autoinoculation or by direct extension.

Pathology. The pathology is characterized by acanthosis, edema, and minute abscesses of the prickle-cell layer. Perivascular infiltration of the papillary layer is also present.

Symptoms. The sites of predilection are the regions of the body which are easily accessible and may result from scratching. The lesions are often linear and are usually multiform, although pustular lesions predominate. In mild cases and in the convalescent stage, the patches are dry, scaly and fissured. In the severe cases, marked edema is accompanied by large vesicles and pustules is present. The primary lesion is a circumscribed patch covered with vesicles and pustules which spread peripherally and may coalesce with similar patches. The skin manifestations are usually pruritic and are often accompanied by urticaria.

from the disease thus suggesting a toxic or neurogenic origin. Cases are recorded of patients, who after suffering attacks of herpes gestationis during several pregnancies continued to develop the eruption after delivery as typical dermatitis herpetiformis. Eosinophilia is usually present just as in dermatitis herpetiformis. Testing of the skin however with iodides or bromides gives negative results. In severe cases, termination of the pregnancy may have to be considered.

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Prognosis. The prognosis is guarded and complete recovery is rare. The outlook is favorable in children. Spontaneous recovery however does occur. Dermatitis herpetiformis usually continues for years without endangering health. Subjective symptoms can always be relieved.

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- (3) follicular (acneform) reactions;
- (4) ulcerating and vegetating eruptions.

The skin manifestations may result from a single small dose but more often the effect is from a gradual accumulation which exceeds the threshold of tolerance that exists in all persons. Not infrequently a state of hypersensitivity is created by intermittent doses, so that a small dose followed by a long

in the extremities, tinnitus, rhinitis, pharyngitis, hyperpyrexia, etc.

After the drug is discontinued, the skin lesions disappear and are usually gone in the course of weeks. The granulomatous formations produced by bromides and iodides slowly involute and may persist for months. Hyperkeratosis and pigmentation caused by arsenic last indefinitely and have a tendency to be



Fig. 189 Dermatitis Medicamentosa. Right: Generalized erythematous eruption without subjective disturbances.

free interval and repetition of the dose produces a skin eruption which did not occur after the first administration. When the eruption appears after a single dose, there may be an interval of several hours between the ingestion of the drug and the cutaneous manifestations, the interval being short in those who are highly susceptible. Erythematous eruptions usually appear sooner than the other types. The skin eruption may be associated with general symptoms of intoxication, such as nausea, cramps, diarrhea, pains

come more severe as time goes on. Arsenical eruptions may not appear for years after the administration of arsenic has been stopped. Fixed eruption due to phenolphthalein may last a long time after cessation of the drug.

Diagnosis. Drug eruptions are distinguished from other skin conditions, which they may simulate, by their sudden appearance; by atypical distribution, usually symmetrical and widespread by a brighter color and by the absence of systemic disturbance. The

Diagnosis It is differentiated from eczema by the marked definition of the areas and the fact that the vesicles are larger than those seen in vesicular eczema.

On microscopic examination it is differentiated from ringworm by the absence of spores and mycelial threads.

Prognosis Proper treatment produces a cure; this disease responds to treatment; however recurrences are not infrequent.

Treatment Application of a 5 per cent *sulfathiazole* ointment or a 5 per cent *ammoniated mercury* ointment is usually curative. Injections of *autogenous vaccine* or of *foreign proteins* are serviceable in preventing recurrences. *Hot dressings* of an aqueous solution of 1/5000 permanganate of potash or an aqueous solution of 1/8000 bichloride of mercury are also favorable modes of treatment.

Dermatitis Medicamentosa

SYNONYMS: *Drug eruption*,
dermatitis toxica.

Dermatitis medicamentosa is a term applied to skin eruptions caused by the internal action of drugs administered orally, parenterally or by cutaneous absorption.

This group of skin affections is considered separately from those eruptions produced by external applications of chemical irritants; for example, formalin dermatitis and iodoform dermatitis to which the term "*dermatitis venenata*" is applied.

There are over 200 drugs in common use which are apt upon consumption to produce skin eruptions. They cause practically every type of lesion. The same drug may give rise to different manifestations in the same individual; consequently the skin lesions may be uniform or multiform; however the for-

mer is the more common. Drugs unrelated chemically may produce similar lesions, or when combined with other drugs the skin manifestations may be altered. For instance, urticaria may be produced by iodides, bromides, any of the analgesic or hypnotic coal-tar products, phenolphthalein, arsenic, quinine, jaborandi, copaiba, tolu, etc., whereas the iodides may also cause papules, pustules, bullous lesions, nodules, petechiae, or fungating growths. The skin manifestations may be widespread or limited to certain areas, such as the face, wrists or lower legs, and are often symmetrically distributed.

Incidence Sutton and Sutton state that dermatitis medicamentosa occurred in about 1 of 2000 patients seen in the University of Kansas Hospital. The large majority were due to arsenical antiluecic remedies and in this group fatalities occurred.

Abramowitz (1938) reported an incidence of 0.15 per cent in 58,000 cases seen in the New York Skin and Cancer Unit.

Symptoms The eruptions produced by drugs, while not sufficiently characteristic to indicate the causative agent, are usually distinctive enough to suggest a medicinal cause. The diagnosis is established by careful questioning and, when necessary, by testing the patient's sensitiveness to the drug by giving a dose of it which will cause an exacerbation of the skin lesions if it has an etiologic relation.

The eruptions fall into four general categories: (1) vascular reactions (erythema, urticaria, angioneurotic edema, purpura, erythema nodosum, erythema multiforme, fixed circumscribed, erythematous and pigmented eruptions); (2) eczematous reactions, such as erythema, vasculation, exudation, or scaling.

usually with intense edema. Lesions may also consist of localized erythematous, urticarial, and herpetiform patches or fixed erythemas.

Antimony rarely produces an erythematous or urticarial eruption. Puente reports a fixed pigmented eruption from tartar emetic.

Antipyrine frequently produces skin eruptions which are of the erythematous type, occasionally morbilliform, scarlatiniform, or fixed erythemas. Urticarial, pustular and bullous lesions have also followed the use of antipyrine and they often occur on the mucous membranes. The eruptions may be followed by desquamation and pigmentation. Moderate itching may be present.

Antivenom eruptions usually follow the injection of animal serum. Serum (animal) commonly causes urticarial or erythematous eruptions. They may be morbilliform or scarlatiniform or may resemble erythema multiforme. Such manifestations are caused occasionally by injections of antitoxins, most often of diphtheria, tetanus, or erysipelas. Usually the eruptions are accompanied by fever, generalized adenopathy and effusions into the joints. The favorite sites are the thighs and buttocks. Bullae and even hemorrhages may be present. Often there is an associated edema of the eyelids, hands, genitals, and feet. Serum eruptions may occur from one to thirty days after injection, more often between the sixth and the tenth days. Attacks last from a few hours to a week; however the erythema multiforme or purpuric types may persist for many months.

Aprot (ergoaprot) may produce erythema, gingivitis, buccal gangrene, edema of the vulva, methemoglobinuria, nephritis, and anemia.

Arsenicals: Arsenic frequently causes skin eruptions. These may be due to

hypersensitiveness and occurring after a few doses, or they may be the result of long-continued use of the drug. The former are widespread erythematous, bullous, papular or pustular eruptions, or even an extensive generalized exfoliative dermatitis, accompanied by constitutional symptoms, such as abdominal pain, diarrhea, pains in the extremities, fever and edema, especially of the fingers, eyelids, and feet. The slow action of long-continued administration may not be manifest clinically for a decade or more after the drug has been stopped. Palmar hyperhidrosis is a common symptom. The late lesions due to arsenic consist of epithelial hyperplasia with the formation of keratoses, occurring chiefly on the palms and soles, upon which carcinoma frequently develops. The keratosis occurs as a discrete, warty lesion, or in a diffuse form, and has follicular tendencies, being especially prominent around the sweat pores. The keratosis is distinctive, usually being symmetrical and surmounted by small black, shiny plugs of keratin. Arsenic is present in the keratoses, being distributed between the cells of the horny layer and also in the cornum of the normal skin surrounding the keratoses. Fissures frequently are observed and may likewise be the site of carcinoma. A follicular keratosis resembling keratosis pilaris may occur in a widespread manner, being especially pronounced over the chest, where it may also involve the nipples. Recurrent conjunctivitis has been reported by Millian as resulting from long use of arsenic.

Arsenical carcinoma may not appear for several years after the drug has been stopped. These carcinomas are usually of the squamous-cell variety; however both basal-cell lesions and those with adenoid structure may occur in the same case and presumably from the same

course of the drug eruption does not follow that of any of the exanthemata. Patients with a suspected drug eruption should always be asked whether they have taken any headache or "stomach" medicine or laxatives. Some of the compounds noted are also present as coloring and preservative agents in cakes, candy and other foods.

Practically all drugs may cause erythematous and urticarial eruptions, which may be regarded as vascular reactions. Macular erythematous eruptions are usually widespread and involvement of the trunk, arms, and face may be morbilliform or scarlatiniform in character. The erythema multiforme type of drug eruption is very common particularly from iodides, while the fixed type of eruption is rare.

Urticarial eruptions may be produced by the same drugs that produce erythematous eruptions.

Vesicular eruptions are rarely caused by the internal administration of drugs. An eruption simulating herpes zoster and herpes simplex may be produced by the administration of arsenic.

Bullous eruptions are produced by iodides, quinine, antipyrine, phenolphthalein, luminal, the salicylates, the bromides, and chloral, being especially common when the last two are mixed.

Pustular eruptions are produced by bromides and iodides, antimony, antipyrine, arsenic, chloral, salicylates, and turpentine. These are most commonly acneiform (bromides, iodides, chloral) but may be vegetating granulomas (bromides and iodides) or ulcerations with gangrene (arsenic, chloral, bromides, iodides, and quinine).

Hemorrhagic lesions occur with the formation of ecchymoses or purpura following the use of such drugs as *copaiba*, ergot, bromides, and iodides, qui-

nine, antipyrine, salicylates, sulfonal, and often animal serum. Keratotic lesions and pigmentary changes are both produced by arsenic. Pigmentation is also caused by the silver salts, particularly silver nitrate, silver arsphenamine, and by bismuth.

Fixed eruptions are circumscribed lesions that persist or recur at the same site over a period of months or years. Erythema perstans with pigmentation caused by phenolphthalein is a classical example. The fixed eruption is essentially a sensitization phenomenon. The lesions usually react violently to small test doses of the drug. Fixed eruptions may follow the use of phenolphthalein, antipyrine, amidopyrine, sodium alurate, arsphenamine, neoarsphenamine, silver arsphenamine, mapharsen, tryparsamide, antimony and potassium tartrate, bismuth, cinchophen, potassium iodide, hypodermic iodine, ipecac, isacrin, mercury, quinine, acetylsalicylic acid, sodium salicylate, scammonium and tryptamine.

Eruptions Produced by Common Drugs. *Acetanilid* usually produces a widespread blotchy erythematous eruption. Large doses of acetanilid may cause cyanosis, which is a symptom of methemoglobinemia.

Acetylarsen may produce an erythematous, pigmented or fixed type of eruption.

Acetophenetidin causes erythematous, urticarial, erythema multiforme, and circumscribed edematous fixed types of eruptions.

Aconite may cause erythematous or vesicular eruptions.

Acriflavine has been reported to cause a repeated urticarial and erythematous eruption in the vicinity of the site of injection.

Amnopyrine frequently causes eruptions of the erythema multiforme type.

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Acetylarsen may produce an erythematous, pigmented or fixed type of eruption.

Acetophenetidin causes erythematous, urticarial, erythema multiforme, and circumscribed edematous fixed types of eruptions.

Aconite may cause erythematous or vascular eruptions.

Acriflavine has been reported to cause a repeated urticarial and erythematous eruption in the vicinity of the site of injection.

Aminopyrine frequently causes eruptions of the erythema multiforme type.

in the soles may even precede this. The skin is hot to the touch and intensely edematous. There is marked swelling of the face and legs. Some cases are mild and disappear without constitutional symptoms. In others, a rise in temperature occurs, ranging from 102 to 103 F. The exfoliation often continues over a period of months. During this period, furunculosis is likely to develop and foci of infection from which the patient suffered prior to the onset are likely to become active. The loss of sleep due to itching and the loss of weight due to fever, restlessness and low intake of food gradually produces marked malnutrition. The pulse is rapid, the mouth parched, the tongue swollen and coated, bronchitis and nephrosis usually complete the picture. Death results from bronchopneumonia, abscess in the viscera, hemorrhage, or uremia. In patients who survive the hair and nails fall out. A brownish pigmentation often remains for several months, and hair and nails are the last to return to normal.

Milian has called attention to the "ninth day erythema" which is a morbilliform or scarlatiniform eruption that appears usually on the ninth day after the first injection of an arsenical preparation. There is no tendency to scaling or vesiculation and the condition is self-limited, disappearing in spite of continued arsenphenamine therapy and without any sequelae. "The ninth day erythema" is characterized (1) by occurring almost without exception nine days after the administration of the first injection of the drug, and (2) by the short duration of the eruption. The eruption is generally morbilliform somewhat resembling a drug eruption. It is differentiated from measles in that it seldom first appears on the face and neck, and by the absence of Koplik's spot on the

mucous membrane or coryza. The mucous membranes of the throat may be congested and there may be nausea or vomiting. The fever is usually not high, although it may reach 103 F. Sometimes a rise in temperature precedes the eruption. The temperature falls rapidly and returns to normal in three or four days. Clandular involvement, particularly of the cervical groups, may be present. Erythema of the ninth day is more common in adults, particularly in women.

Postarsphenamine dermatitis is preventable in most instances. The condition is most apt to occur during the first course of arsenphenamine injections, particularly in those receiving simultaneous arsenical and heavy-metal therapy.

OXOPHENAMINE HYDROCHLORIDE (ALAPHASEN) This may cause erythematous and fixed types of eruptions and exfoliative dermatitis.

TRYPARSAMINE: This arsenical may cause pruritus, urticaria, exfoliative dermatitis, and fixed eruptions. Optic atrophy has also followed its use.

TREATMENT IN ARSENICAL DERMATITIS The treatment is mainly supportive. The diet should be low in protein content and consist of plenty of starch foods, butter, milk, gruels, cereals, and vegetables. Dextrose water should be given freely by mouth and a 10 per cent solution of dextrose intravenously is perhaps more valuable than anything else for the relief of itching, exudation, and inflammation. Injections of liver extract cause general improvement. Sodium thiosulfate is given both intravenously and by mouth, 1 gm. daily (by both methods in the beginning) and gradually increased to 2 gm. daily by vein, the latter being divided in doses of 1 gm. morning and night, which should be continued for about a week. This drug has a tend

cause Lesions on the trunk are generally of the superficial flat variety and may simulate Bowen's disease. On the hands and feet arsenical carcinoma is more likely to be of the cornifying type, at first producing a horny papillomatous growth which later metastasizes with or without preceding ulceration.

Pigmentary deposits frequently follow the long-continued use of arsenic. The patches may be small and somewhat confluent so that there is a mottled appearance or the patches may be large. The color is brownish or sepia with a glistening sheen.

The pigmentation is accentuated in dark skinned individuals and on parts of the body which are ordinarily pigmented such as the areolae and are subject to pressure from clothing as the sides of the neck. When the arsenic has been given for the treatment of some skin disease the pigmentation is likely to be pronounced at the sites of the previous lesions. Arsenical pigmentation does not occur on the mucous membranes. It disappears after the administration of the drug has stopped the disappearance being hastened by the intravenous use of sodium thiosulfate solution. The distribution is more limited than is observed in Addison's disease in this affection there is usually an involvement of the mucous membranes.

Hyperidrosis commonly precedes or accompanies the development of hyperkeratosis due to arsenic occurring chiefly on the palms and soles.

Herpes zoster sometimes occurs after the use of arsenic, being due either directly to the drug or to a predisposition caused by the drug to infection by the herpes virus. The production of arsenical neuritis and optic atrophy from the use of some arsenical compounds has also

been observed and arsenic has also caused herpes simplex both on the face and on the genitals. The nails become thickened and brittle and may be shed, due to the action of arsenic upon the matrix. Such changes are usually associated with hyperkeratosis of the palms and soles.

ARSPHENAMINE. Arspenamine rarely causes typical arsenical eruptions, but there are other manifestations which are more distinctive. The combined use of mercury and arsenic may account for the cutaneous sensitiveness to arspenamine, the failure to eliminate the arsenic due to renal impairment from mercury being responsible for the skin reaction. Arspenamine dermatitis is apparently due to a sensitization of the skin. Contributory factors are other skin diseases, focal infection and anemia.

The skin reaction to arspenamine may be observed after the first injection or at any time thereafter and in some instances there seems to be a hypersusceptibility created by a long rest interval between injections. The mild eruptions are usually erythematous, scarlatiniform, morbilliform, urticarial, and eczematous, and may be followed by desquamation. These are generally of short duration and apparently due to anaphylaxis, being in the same category as the nutritoid crises. Herpetic eruptions and lichenoid eruptions are also encountered sometimes fairly characteristic of lichen planus or lichen spinulosus. Paroungian states that pigmentation is accentuated in Negroes following treatment with arspenamine.

Arsenical exfoliative dermatitis usually begins on the extremities as an itching eczematous or maculopapular eruption with slight scaling and becomes widespread and exfoliates in the course of a week. Occasionally tingling

the genitals are characteristic of phenobarbital therapy.

BARIUM SODIUM. This may provoke erythematous, urticarial, papular vesicular or bullous eruptions. Morbilliform and scarlatiniform manifestations occur and there may be erosive oral lesions.



Fig. 191 Bromoderma. Of legs. Note crusting character at borders.

Benzoic acid and *sodium benzoate* may produce erythematous or papular eruptions.

Bismuth produces a slate-colored line upon the gums and ulcerative stomatitis and has been credited with the causation of exfoliative dermatitis and herpes sceler. It also causes pruritus, urticaria, erythema bullous, or hemorrhagic lesions. Lichenoid, papular eczematous eruptions and conjunctivitis have been reported. Embolism, ulcerative colitis, agranulocytosis, arthralgia, and nephritis have also been reported following the use of bismuth. Sodium thiosulfate, parenterally or orally is the most effective treatment.

Borax and *boric acid* may cause erythematous, papular scaly or multiform

lesions, or produce edema of the eyelids and conjunctivitis.

Bromides commonly produce skin eruptions which are fairly distinctive. They may occur after a small dose or after protracted use of the drug, and they may be seen in children the drug being transmitted by the milk of nursing mothers. The commonest lesions are acneiform with pustules of a rather inflammatory type which are observed not only on the face, but on parts of the trunk and in hairy regions. The lesions are frequently vesicular and may fuse to form large patches which become suppurative, crusted, or vegetative. These are soft and occur most often on the legs. Bullous lesions as well as tender nodules also appear.

TREATMENT IN BROMIDE DERMATITIS.

Wile, Wright, and Smith demonstrated that there was a ready substitution of chloride for bromide in the body fluids, and suggested the use of salt solution intravenously in cases of bromide eruption. A certain amount of shock may develop immediately after the injection and also a transient nephritis. They used from two to five injections of from 100 to 400 cc of deionized saline solution intravenously at intervals of three or four days. If the patient takes large quantities of table salt by mouth, the same results are achieved, though less rapidly.

Cannabis indica may cause a pruritic papulovesicular eruption or a transient pigmentation.

Cantharides may produce an erythematous papular or vesicular eruption. The lesions are more prone to occur on the genital regions.

Capricorn may produce an itching, erythematous eruption. This is very rare.

Carbromal often causes erythematous, macular or urticarial eruptions.

ency to produce alkalosis so it should not be continued indefinitely. Dimer capto dithiopropanol or dimercaprol (B.A.L., or British antilewisite) used as a solution in peanut oil and benzyl benzoate and packaged in sterile ampules containing 500 mg in 5 cc., seems likely to supersede all other remedies for arsenical dermatitis as well as for arsenical over dosage and arsenical encephalitis. It must be given early and intramuscularly. In severe cases the recommended dose is 3 mg per kg of body weight repeated six times at four hour intervals during the first two days four times daily on the third day and twice daily for the following ten days. In mild cases, 2.5 mg per kg is adequate for each dose (Eagle). Minor but transient toxic reactions may occur (nausea generalized aches, and a burning in the mouth and eyes). The drug removes the arsenic from the tissues by forming a compound with it which the body can excrete. Calcium therapy is also of value. The gluconate may be given by mouth intramuscularly or intravenously. Blood transfusion is recommended by Dennis. Roentgen therapy relieves the itching and diminishes the inflammatory condition, however sedation is usually necessary.

Atropine or belladonna usually produces scarlatiniform eruptions or isolated erythematous patches which may be accompanied by severe itching. Children are especially susceptible to belladonna. Pyrexia and convulsions may follow the use of atropine.

Barbiturates BARBITAL. Barbitol produces urticarial erythematous, and bullous eruptions, which may also simulate erythema multiforme. Numerous hypnotics of the barbituric acid series cause similar skin eruptions, and those that contain antipyrine, aminopyrine and alurate produce the localized fixed type

of erythema. Pruritus, purpura, leukoplakia and photosensitization may also be caused by the use of barbitol.

PHENOBARBITAL. This causes generalized pruritus and jaundice associated



Fig 190: Generalized Morbilliform "Dermatitis Medicamentosa Interna." Due to phenobarbital.

with erythema urticaria morbilliform eruptions, and bullae. Fever may accompany the eruption and hyperpigmentation may follow it. Bullous and erosive lesions in the mouth and on



Fig. 192 Seborrheic Dermatitis. (Courtesy of Dr. Carroll S. Wright.)

Chloral frequently causes eruptions which may be scarlatiniform erythemas or localized erythematous patches papules, bullae and pustules. Some of the lesions may be hemorrhagic and at times there is pyrexia. These eruptions may be accompanied by cyanosis of the extremities. Pruritus is occasionally present.

Chloralamide and *chlorobutanol* produce lesions similar to chloral although much milder.

Chloroform may provoke an erythematous or purpuric eruption.

Cinchophen usually produces angioneurotic edema, urticarial and erythematous eruptions and may produce eruptions simulating erythema multiforme.

Codeine causes pruritus and at times extensive erythema which may be followed by desquamation.

Copaiba frequently produces urticaria, erythematous or bullous lesions associated with intense itching or burning. The lesions may be followed by desquamation. At the onset there may be sore throat and constitutional symptoms, including fever.

Cubeb produces lesions similar to *copaiba*.

Digitalis may cause erythematous, scarlatiniform and papular eruptions, which may be followed by desquamation.

Dinitrophenol has been known to produce pruritus, maculopapular erythema, urticaria, edema, purpura and dermatitis exfoliativa. Cataracts have also been reported following the use of this drug.

Ephedrine produces erythematous, morbilliform and eczematous eruptions, sometimes urticaria or purpura. Lesions produced by ephedrine often simulate pompholyx.

Ergot may cause bullous, pustular or gangrenous lesions. Similar conditions

may be produced by ergotamme tartrate. Papaverine is the antidote for ergot poisoning.

Eucalyptus may cause erythematous and urticarial eruptions.

Fluoride may produce an eruption not unlike pustular psoriasis. Mottling of the teeth occasionally follows its use.

Gold produces erythematous, urticarial and exfoliative eruptions. Melanoderma and keratoderma have also been reported following gold therapy. *B.A.L.*, or dimercaprol, has been used as an antidote (see Arsenic p. 286) with success.

Guaiacum causes erythematous and urticarial eruptions.

Hyoscyamus may produce erythematous eruptions.

Insulin may produce urticaria, pruritus, and morbilliform erythema, also tumefaction and lipodystrophy in and around the sites of injection. Protamine insulin may produce allergic manifestations in patients sensitive to protids.

Iodides commonly cause acneform eruptions with numerous small follicular pustules, each surrounded by a ring of hyperemia. Acneform eruption and exacerbations of acne vulgaris are often due to iodized table salt. They have also been seen in patients on prolonged thyroid medication. Bullous lesions from iodides are also common and may become ulcerative and crusted. Vegetative or fungal lesions are occasionally encountered and may be mistaken for true neoplasms. Lipoiodine has been responsible for recurrent painful folliculitis of the nostrils. Purpura and urticaria are common manifestations of mild iodism. The eruptions also may be furuncular, carbuncular or of the erythema multiforme or erythema nodosum type. Malignant and fatal types of iododerma may ensue after the administration of very small amounts of iodides, often much less than the usual

therapeutic dose. Treatment is the same as that described for eruptions due to bromides.

Iodoform may produce macular papular and bullous lesions.

Ipecac and *emetine* have produced local pruritic, edematous, erythematous, and vesicular patches.

Iron according to Sequeira, has produced acneiform pustules.

Scarlatiniform eruptions are the most common. In addition to the skin manifestations, mercury produces a stomatitis. Goeckerman has reported the use of a cosmetic containing calomel. Fixed eruptions that are either erythematous, pigmented, or vesicular have been ascribed to injections of calomel bichloride, and other mercurial compounds.



Fig. 193 Dermatitis Medicamentosa. Left "Fixed" eruption. Right "Fixed" eruption (phenolphthalein)

Jaborandi and *pilocarpine* rarely may produce urticarial and erythematous eruptions.

Liver extract may produce erythema, urticaria, and asthma.

Methenamine may produce localized erythematous lesions or generalized morbilliform eruptions.

Mercury infrequently causes erythematous and scarlatiniform eruptions or rarely a generalized exfoliative dermatitis. The eruptions may resemble folliculitis, erythema multiforme, urticaria, purpura, measles, and pemphigus.

Dimercaprol (in a dose of 5 mg per kg of body weight, given in the same manner as for arsenic poisoning) together with the usual supportive measures, has been found of benefit in mercurial poisoning.

Morphine and *opium* may cause erythematous, morbilliform, scarlatiniform, and urticarial eruptions which itch intensely and usually desquamate.

Nicotinic acid may produce transient vasodilatation which is characterized by a feeling of warmth, tingling, flushing, itching, and urticaria. Dizziness, nausea, and vomiting have been reported.

ACETYSALICYLIC ACID This rarely produces cutaneous lesions, although erythematous, papular and hemorrhagic eruptions, as well as urticaria, angioneurotic edema, and exfoliation have been described. Hypersensitivity to as-

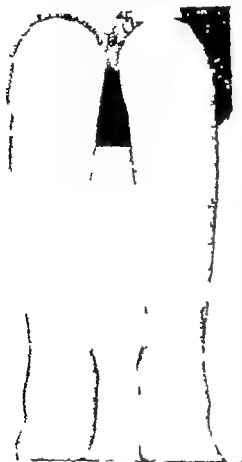


Fig. 198 Erythema Nodosum. Following ingestion of sulfadiazine.

pirin is the most common form of drug allergy symptoms of which may be either asthma, urticaria, or angioneurotic edema. Recurrent erythema multiforme and purpura have also been reported.

SALOL. Salol may cause blotchy erythematous eruptions.

Santonin may cause erythematous and morbilliform eruptions.

Silver or its salts, after prolonged use, may produce a metallic blue discoloration of the skin (argyria) either generalized or localized.

Sodium thiosulfate may produce a purplish or vesicobullous eruption, or generalized exfoliative dermatitis.

Sulfanilic acid may cause erythematous eruptions, urticarial, papular and morbilliform or scarlatiniform erythemas and photosensitization. These eruptions are more likely to occur on the parts of the body exposed to sunlight. Pruritus, purpura, cyanosis, and optic neuritis have also been reported following its use. Porphyrinuria and acute yellow atrophy have also been reported.

Sulfathiazole occasionally produces papulopustular lesions which simulate varicella.

Sulfadiazine may cause erythematous (especially macular) and fixed eruptions, bullous and purpuric lesions, and erythema nodosum.

Sulfonal rarely produces erythematous and scarlatiniform eruptions.

Turpentine may cause erythematous, pruritic, papular and morbilliform eruptions.

Prophylaxis Prophylaxis consists in the avoidance of the drugs known to be causative of the eruption.

Treatment Treatment, in general, consists in stoppage of the drug which the patient may be taking. Specific therapy where possible has been indicated.

Dermatitis Menstrualis

SYNONYM *Dermatitis dysmenorrhoeica*.

Patients suffering from dysmenorrhea, amenorrhea, oligomenorrhea, or other dysfunctions of the ovarian system are

Penicillin Toxic reactions, due to this antibiotic or to its impurities in commercial preparations result both from its local and parenteral use and are increasing. Contact dermatitis, as a result of primary irritation and sensitization, has been observed. Primary shock reactions and sensitization reactions from its paren-



Fig. 191 Dermatitis Medicamentosa. Following three day use of sulfathiazole

teral use have been observed. Among these are pruritus, erythemas, urticarias, giant urticarias, generalized dermatitis, and even exfoliative dermatitis. Focal reactions (Jarisch Herxheimer reactions, the therapeutic shock and paradox) are frequent in acute but also occur in chronic syphilis. The antihistaminics (benadryl and pyribenzamine) appear to be specifics for the allergic reactions. They may also be given prophylactically to patients known to be sensitive but requiring penicillin therapy. Flippin states that penicillin used locally may sensitize patients to its parenteral use.

Reactions from penicillin (and serum

sickness) have been successfully combated by the slow intravenous drip of 1 gm (15 grains) of procaine hydrochloride in 500 cc of isotonic sodium chloride solution (State and Wangersteen).

Phenolphthalein produces bullous and erosive lesions in the mouth and on the genitals. Fixed lesions which are persistent smooth macular plaques, varying in size up to several centimeters, may occur. The plaques are polychromatic usually slate colored or violaceous, with ill defined borders. In the course of time, the color changes and the central part becomes depigmented. Generalized erythematous eruptions resembling measles, scarlet fever or erythema multiforme may also occur. Universal pigmentation has also been reported.

Phosphorus causes bullous and purpuric lesions.

Potassium sulfocyanate and other sulfocyanates used for hypertension often produce generalized exfoliative dermatitis.

Procaine may produce an erythematous and vesicular eruption which is very pruritic.

Quinine not uncommonly produces a scarlatiniform erythema or general urticaria. The following types of eruption have also been reported: erythema, purpura, bullae, eczematous lesions, all associated with severe itching and often followed by desquamation. Gangrene has followed the use of quinine and urea hydrochloride. Abramowitz and others have reported fixed erythema from quinine.

Salicylates Salicylates and salicylic acid rarely cause erythematous, urticarial or bullous lesions. Eruptions may be scarlatiniform or morbilliform and followed by profuse desquamation of the epidermis.

ACETILSALICYLIC ACID This rarely produces cutaneous lesions, although erythematous, papular and hemorrhagic eruptions, as well as urticaria, angioneurotic edema, and exfoliation have been described. Hypersensitivity to as-

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Sulfonamides may cause erythematous eruptions, urticarial papular and morbilliform or scarlatiniform erythemas and photosensitization. These eruptions are more likely to occur on the parts of the body exposed to sunlight. Pruritus, purpura, cyanosis, and optic neuritis have also been reported following its use. Porphyrinuria and acute yellow atrophy have also been reported.

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Dermatitis Menstrualis

SYNONYM *Dermatitis dysmenorrhoealis.*

Patients suffering from dysmenorrhea, amenorrhea, oligomenorrhea or other dysfunctions of the ovarian system are

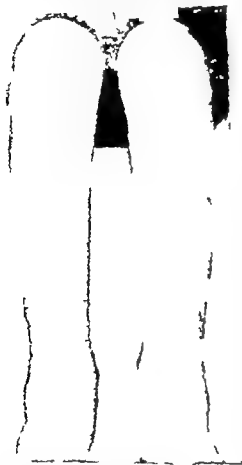


Fig. 195 Erythema Nodosum. Yellowing, ingestion of sulfadiazine.

pirin is the most common form of drug allergy symptoms of which may be either asthma, urticaria, or angioneurotic edema. Recurrent erythema multiforme and purpura have also been reported.

Salol. Salol may cause blotchy erythematous eruptions.

often subjects of a dermatitis. The eruption is characterized by symmetrically located erythematous patches, urticarial wheals and occasionally nummular bullous or herpetic areas of dermatitis. The sites of predilection are the extremities and face. The eruption may appear periodically. It often occurs one or two years prior to menstruation. Vulvar pruritus is often an associate symptom.

Varieties Two varieties are described. The first type is of neurogenic origin, while the second type is an infectious dermatosis (pustular bacterid).

Neurogenic Dermatitis Repens

Incidence It is a common affection of the skin.

Etiology Dermatitis repens often follows an injury or a preexisting derma-

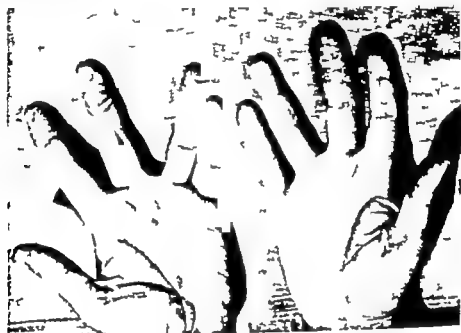


Fig. 196 Acrodermatitis kerstan (Hallopeau) (Courtesy of Dr. Carroll S. Wright.)

Treatment *Estrin* given intramuscularly in doses of 1 mg. three times a week or thyroid extract in small doses is helpful and occasionally curative.

Dermatitis Repens

SYNONYMS: *Acrodermatitis con tinua* (Hallopeau) *acrodermatitis perstans*, *recurrent phlyctenoses of the extremities* (Audry)

Dermatitis repens is a chronic inflammatory dermatosis which is characterized by the undermining of the upper layers of the epidermis with numerous minute pustules.

tosis (dyshidrosis) or it may appear spontaneously. Dermatitis repens is perhaps a form of pustular dyshidrosis. The organisms that may be present are regarded as secondary invaders.

Pathology The primary lesion occurs in the epidermis and is characterized by subcorneal abscesses, formed in the upper layer of the rete. The abscess finally dries and exfoliates with the overlying parakeratotic horny layer.

Symptoms It usually begins on the finger and palms or on the toes and soles either as a pustule or as paronychia. At the onset the above condition is unilat

eral and localized however within a few hours to a few days vesicles or bullae appear. The patient does not complain of the feeling of tension or pain which is common to cutaneous infections. New pustules present themselves adjacent to its predecessors and in this manner vari-

may atrophy and the underlying osseous structure become involved. The disease seldom becomes generalized or spreads to distant parts, occasionally the mucous membranes of the mouth are involved. Mouth lesions consist of fissures or circular plaques surrounded by inflammatory



Fig. 197 *Acrodermatitis Parvula*. Left Of ten years duration before treatment with salicypyridine. Right After seven weeks treatment with salicypyridine (2 gm. daily) (Courtesy of Dr. Walter F. Lever.)

ously sized plaques develop. The vesicles rupture and the epidermis is destroyed, leaving a raw surface or there may be fine branny silvery scales.

Involvement of the nail bed may result in the presence of vegetating lesions. The nail may eventually be entirely lost. If the condition is present for a long period of time the fingertips

areolae. The tongue may also be affected and be covered with a diphtheroid membrane.

Slight itching or a burning sensation constitutes the subjective symptoms of the cutaneous lesions, while the mucous membran lesions are quite painful.

Diagnosis A history of recurrences of the pustular lesions and their resist-

ance to treatment are characteristic of the disease.

Prognosis. *Dermatitis repens* may persist for months or years and the outlook is therefore guarded. It is very resistant to treatment. If treatment is, however, started early, the chances for recovery are good. If atrophic changes have already occurred, nothing can be done.

Treatment. Use of a lotion of 1:8000 *permanganate of potash* or crude coal tar ointment with 5 per cent ammoniated mercury is helpful in the early stages. The constant application of a 1 per cent solution of *potassium permanganate* has been used with benefit. *Staphylococcus toxoid* is occasionally beneficial. *Sulfapyridine* has been of value in some cases.

Pustular Bacterid

The second type of *dermatitis repens*, or pustular bacterid, is an infectious dermatosis. The early lesions are intra-epidermal vesicles which soon become pustules and occur in single foci on the plantar or palmar surface. It may appear on normal skin or complicate *dyshidrosis* or other cutaneous eruptions. The infection spreads slowly beneath the stratum corneum. The pustules are tense and are surrounded by an inflammatory areola. On removing the epidermis over the lesion, a foul odor may be noted. It often spreads until the entire palmar or plantar surface is involved. Pain and itching in varying degrees may be a subjective symptom.

Etiology. *Staphylococci* are found abundantly in the lesions but are probably not the causative factor.

Prognosis. This type of dermatitis *repens* is apt to recur and it sometimes responds to antiseptic treatment. It is apt to be rebellious to therapy.

Treatment. Foci of infection should be eliminated. Pustules should be opened and dressed with a solution of *potassium permanganate* 1:8000 or *Buro's solution* 1:4 and 5 per cent ammoniated mercury used at night. The oral use of *sulfapyridine* is effective but must be given indefinitely if recurrence is to be avoided. Cataphoresis with copper sulfate solution (> 10 to quart) has been used with success.

Dermatitis Seborrheica

SYNONYMS: *Seborrheic eczema*, *seborrhea corporis*, *seborrhea corporis maformis*.

Seborrheic dermatitis is an inflammatory skin disease characterized by variously sized discoid, oval, circinate, or gyrate yellowish lesions which may be either dry and scaly or greasy and crusted.

Varieties. The disease may be acute, subacute or chronic.

Incidence. The disease occurs most often in patients between the ages of twenty and thirty-five. Both sexes are affected, although it occurs more frequently in males about the age of puberty.

Etiology. The cause is unknown. The *Pityrosporon ovale*, a pleomorphic micro-organism, is demonstrable in most eruptions of *seborrheic dermatitis*. Most investigators regard this organism as a secondary invader and not as the causative factor. Endocrine disturbances, faulty diet, and heredity and environment are contributory factors. Hereditary and familial incidence is often established by the patient's history.

Symptoms. The subjective symptoms consist of various degrees of itching. The scalp, ears, postauricular and genito-crural regions are often the seats of severe itching.

This disease has been erroneously considered as contagious. The sites of predilection are the regions of the body which are most abundantly supplied with sebaceous and sweat glands, i.e., the scalp, forehead, nose, ears, sternal, interscapular, axillary, submammary, umbilical, and genitocrural regions. The flexor surface of the knees and elbows is occasionally affected.

Seborrheic dermatitis usually begins on the scalp and then extends to the face, neck, and trunk. While this is the usual method, occasionally the initial eruptions start on the trunk and extremities. Eruption on the scalp may be either dry and scaly or it may consist of oily yellowish, adherent scales and crusts. The appearance of the former is that of dandruff while in the latter inflammatory symptoms are more or less pronounced. The entire scalp is often affected and the process may extend to the forehead, ears, postauricular regions, and neck. The hair is either dry and lusterless or oily and sticky. In some instances, the scalp eruption appears as isolated, greasy elevated areas of scales and detritus. In more advanced forms, the oily eruptions fuse and cover the entire scalp. When this occurs, an unpleasant odor may be present. Loss of hair may be rapid and profuse or there may be no hair loss at all. Complete loss of hair rarely occurs.

On the neck, the eruption occurs in the form of circinate scaly patches which have a reddish yellow hue.

On the eyebrow, ala nasi, cheeks, and around the mouth, the lesions are reddish yellow in color and are covered with thin adherent scales which cover a more or less inflamed area. The ears and surrounding skin are often edematous, red, and scaly. Fissures, serous exudation, and scaling commonly occur in the post-

auricular region. The auditory meatus is frequently filled with many scales and crusts.

On the forehead and cheeks, a papular form of the eruption often occurs. These lesions are usually the size of a pinhead or lentil and are covered with a yellowish red crust removal of which reveals



Fig. 196 Seborrheic Dermatitis.

an inflamed and moist surface. The disease sometimes affects the hip, where it is called *heilitis exfoliativa*.

If the disease occurs on the trunk, the initial lesion is a punctiform macule which extends peripherally and soon becomes covered with a scale. The individual lesions coalesce to form plaques, some with definite borders, while others merge imperceptibly with the normal skin. The surface of these plaques is covered by grayish or yellow scales, which are usually oily. Sometimes the follicles in special areas, usually sternal and interscapular are involved in which case groups of scale topped or pink,

barely palpable papules constitute the eruption (follicular type)

When the trunk is primarily involved the sternal and interscapular areas are the sites of predilection. Inflamed scalp patches often occur in and around the umbilicus and in the submammary region.

In the axillae the disease appears as a sharply defined erythematous scalp

pityriasis rosea atopic dermatitis, neurodermatitis, ringworm, moniliasis, lupus erythematosus, and syphilis.

Seborrheic dermatitis may be differentiated from other types of dermatitis by its yellow color by the fact that seborrheic dermatitis usually begins on the scalp, by the lack of infiltration of the lesions, and by the greasy scales and



Fig. 199: Seborrheic Dermatitis (follicular dermatitis). Note numerous pinhead-sized papules in pre-sternal area; similar lesions present on posterior surface of chest between scapulae.

and crusted yellowish plaque. These plaques may be covered with a serous exudate and be crusted.

On the forearms and hands, the lesions are often patchy and dry and are covered with adherent scales.

The genitocrural and perineal regions are affected in a manner similar to the axillae.

Involvement of the nails is characterized by fragility, transverse and longitudinal ridging of the nail plate and a yellowish gray discoloration.

Diagnosis. Seborrheic dermatitis must be differentiated from eczema, psoriasis,

crusts. Ordinary forms of eczema more often appear on exposed surfaces of the body. The ordinary symptoms of eczema are erythema, vesicles, pustules, crusting, scaling and crusting associated with itching. Psoriasis is characterized by nongreasy lamellar silvery scales. Psoriasis occurs in the scalp as dry circumscribed scaling patches. Lesions developing on the scratch marks are often observed in psoriasis but not in seborrheic dermatitis. Psoriasis does not produce baldness.

Pityriasis rosea usually begins in the form of a "primary plaque" or "herald

spot, which is located either on the trunk, upper thighs, or buttocks. About ten days after the appearance of the primary patch a more or less generalized eruption appears on the trunk. There is no itching and the face and scalp are rarely involved. The eruption



Fig. 200 Seborrheic Dermatitis.

is dry and the diagnostic lesion has a scaly pinkish border while its center is fawn colored and wrinkled in appearance.

Ringworm of the scalp and body can be determined or diagnosed by microscopic or cultural examination. The greasy appearance of seborrheic dermatitis is always lacking in ringworm of the scalp.

The patches of neurodermatitis are dry scaly infiltrated, inflamed, and lichenified. They are attended with marked itching. The scalp is not involved in neurodermatitis.

Eruptions caused by infection with *Monilia albicans* (oedomycoosis) are quite similar to those of seborrheic dermatitis.

Monilia is more likely to occur in diabetic patients. The eruption of monilia lacks the yellow hue and oily appearance of seborrheic dermatitis and the scalp is usually not involved. When the lesions occur on the submammary region or on the intertriginous areas, microscopic examination of the skin scrapings and culture for the fungus is necessary.

Lupus erythematosus is often confused with seborrheic dermatitis, especially with seborrhea oleosa of the face. The eruption seen in lupus erythematosus is dry bluish red, accompanied by very fine telangiectases and in the later stages by patulous sebaceous ducts harboring adherent scales. The yellow tinge



Fig. 201 Seborrheic Dermatitis.
(Courtesy of Dr. Jacques P. Guéguen)

of seborrheic dermatitis is absent in lupus erythematosus.

Secondary syphilis is diagnosed by a positive Wassermann test, generalized adenopathy mucous patches, condylomata, and primary infection history.

Complications The most frequent complication is a secondary eczematization which follows infection with staphylococci streptococci and occasionally *Monilia albicans*. Baldness occasionally occurs on the scalp axillae and pubic regions.

Prognosis *Seborrheic dermatitis* usually responds favorably to treatment. There is, however, a decided tendency to recurrence. If the patient is in good health relapses are not so apt to recur. Hair loss, which is commonly observed in this disease is usually only temporary.

Treatment Any systemic diseases must be treated. Many of the patients are victims of gastrointestinal disturbances and appropriate treatment for this is necessary.

Obese patients should reduce by diet and exercise. Reduction of fats and avoidance of chocolate rich and highly spiced foods, coffee and alcoholic beverages are advisable. Large doses of vitamin B are often beneficial. Liver given orally or large doses of liver given by injection is advisable. A dose of 5 cc of liver extract may be given intramuscularly every second day. Small doses of thyroid gland are occasionally helpful.

Topical Applications Wet dressings are indicated for acutely inflamed areas. The following wet dressings afford the most relief.

1. Borow's solution diluted with water in the ratio of 1:10 or 1:20.
2. Aqueous solution of potassium permanganate 1:4000.
3. Aqueous solution of boric acid, 5 per cent.
4. Aqueous solution of silver nitrate 0.25 to 0.5 per cent.
5. Aqueous solution of resorcin, 0.5 to 2 per cent.

Wet dressings should be covered with oiled silk or wax paper and should not be permitted to become dry.

After the subsidence of the inflamma-

tory symptoms, the following ointments, pastes and lotions are indicated.

1. Sulfur precipitate, 3 to 5 per cent.
2. Resorcinol 3 to 5 per cent.
3. Hydrarg. ammoniat. 2 to 5 per cent.
4. Hydrarg. oxid. fl. 1 to 5 per cent.
5. Salicylic acid, 2 to 5 per cent.
- Tars. Crude coal tar 2 to 5 per cent.
 Oil rose, 2 to 10 per cent.
 Oil cade 4 per cent.
 Liq. carbonis detergens, 5 to 15 per cent.

For nonscaling noncrusting, erythematous, and subacute areas, calamine lotion with 1 per cent ichthyol is advised. Either of the following prescriptions is useful in this type of case.

I	
Resorcinol	2 per cent
Liquor carbonis detergens	4 per cent
Lotion calamine & F. q.s.	250.0
Sig. Shake and apply every 10 or three hours.	

II	
Liquor Barrow's	30.0
Poly zinc oxide	60.0
Pul. talc	80.0
Glycerine.	45.0
1/4 calcis q.s. ad.	250.0
Sig. Shake and apply with flat brush. To avoid caking, applications of olive or mineral oil should be made once or twice daily.	

If the areas are scaly and crusted, one of the following ointments is indicated.

I	
Hydrarg. ammoniat.	1.0
Acid salicylic	1.0
Ung. rosae q.s. d.	30.0
Sig. Apply three times daily.	
II	
Crude coal tar or lintrial	0.5
Ung. acid boris	10.0
Lanolin q.s.	50.0
Sig. Apply several times each day.	

Different regions of the body require topical treatment which differs in the concentration of the various drugs as well as the kind of vehicle employed. The scalp should be shampooed once or twice a week. Castile soap is perhaps

the best soap to use; however if the scalp is very dry tar soap may be substituted, and, if the scalp is oily sulfur soap is probably better. A mineral or olive dressing should precede the shampoo by at least one hour. George Andrews suggests that the following prescription be rubbed into the scalp the night before shampooing:

Hydrarg. oleatis	40 to 100
Oleores. capivi	50 to 40
Menthol	50
Ol. pta. pumil.	000
Ol. amygd. amar.	001
Petrolat. liquid (light) q. s. ad	500

Following the shampoo one of the following is used once or twice daily

I

Zinc sulfocarbathylate	40 to 50
Resorcinoles	40 to 150
Betrasaphthol	50
Spts. lal rect.	1000
Resorcinol. brom.	001
Aq. distillat. q.	2500

Sec. For dark hair only

If the scalp is too dry the following may be added:

Ol. ricini	40 to 120
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For excessively oily scalps and coarse hair George Andrews advises the following:

II

Liquid formalin, 40 per cent	40 to 20
Acid sulfuric	150
Spts. vini rect.	50 to 150
Acetone	150 to 500
Ol. rose geranium	00
Aq. distillat. q. ad	5000

For women with excessively dry scalp, George Andrews advises a brilliantine to be applied sparingly by means of a hair brush

III

Ol. rosae	2000
Ferrocenic (radical)	001
Ol. violae	000
Spts. lal rect. q. ad	5000

When the scalp is very scaly the following is recommended

IV

Hydrarg. ammoniat.	20
Acid sulfuric	20
Ol. ricini	10
Liq. rose q.	500
Sec. Apply to scalp daily for three days, then twice weekly	

After the disappearance of the scales and inflammation, the following lotion may be applied instead of the ointment.

Chloral hydrate	40
Menthol	100
Hydrarg. chlor. corros.	00
Acid sulfuric	50
Ol. ricini	50
Spts. lal rect. 70 per cent q. s.	1000
Sec. Apply by gentle massage daily using finger-coat.	

For intertriginous or flexural areas, shake lotions are preferable to creams or ointments. If lotions are too drying, calamine liniment may be used or temporary compresses of mineral oil applied once or twice daily.

Lemons appearing on the face and neck are usually very pruritic and the following ointment is invariably helpful

Phenol	0.15
Nafalac.	100
Ung. zinc oxide. q. s.	200

Crusted lesions are best treated by applying once a week the following:

Menthol	20
Carbonol	50
Carbon tetrachloride	500

Sec. This should be applied the night preceding the morning shampoo.

Blepharitis responds favorably to a 1 or 2 per cent aqueous solution of brilliant green. It should be painted on the margin of the eyelids twice each day.

The following ointments are equally valuable as and less objectionable than colored medications.

1. One per cent yellow oxide of mercury ointment in petrolatum.
2. Ointment of phenyl boracoric nitrate, 1:5000.
3. Five per cent salicylic acid in equal parts of ung. aquaphor and greasiness cream.

Inflammation of the eyelids usually responds favorably to repeated application of Burow's solution (1:20) boric acid solution (3 per cent) bichloride of mercury (aqueous solution of 1:5000)

In recalcitrant cases, 3 per cent hydroargyrum ammoniatum ointment or 5 per cent sulfathiazole ointment should be tried

Generalized seborrheic dermatitis requires hospitalization. During the inflammatory period of the eruption wet compresses of 2 per cent resorcinol lotion or prolonged baths of the same or a bath consisting of potassium permanganate 1:5000 or colloid baths are very soothing and helpful. After the acute symptoms have subsided shake lotions, such as calamine lotion with 1 per cent ichthylol are advisable.

Mild erythema doses of ultraviolet light given every five to seven days, are a valuable adjunct to treatment. The Gren rays are even more helpful.

Röntgen treatment is necessary for patients who are unable to use topical applications or whose lesions are resistant to topical applications. Forty to seventy-five r doses of Röntgen rays given once a week to affected areas usually give prompt and favorable results. The total dosage should not exceed 200 r to any one area.

Dermatitis Vacciniformis Infantum

Dermatitis vacciniformis infantum is an ulcerating streptococcal infection of the skin of children. The infection is characterized by tiny erythematous macules occurring upon the buttocks and genitalia. The macules soon become umbilicated vesicles filled with clear serum which becomes purulent. The lesions of dermatitis vacciniformis infantum resemble varicella.

Treatment Treatment consists of the application of soothing lotions such as a 1 per cent aqueous solution of aluminum acetate or saturated boric acid solution.

Dermatitis Vegetans

SYNONYMS: *Pyoderma vegetans*,
pyodermatitis vegetans

Dermatitis vegetans is an inflammatory dermatosis characterized by vegetating granular plaques which vary in size and distribution.

Varieties Dermatitis vegetans often resembles pemphigus vegetans, while other cases are not unlike a blastomycetic dermatitis or a bromide eruption.

Incidence Dermatitis vegetans may occur at any age, although it probably occurs more frequently among infants.

Etiology Dermatitis vegetans results from infection of patches of chronic eczema with staphylococci, usually the *Staphylococcus aureus*.

Pathology Masses of exuberant granulation tissue, excess of fibrous tissue and increased vascularity with the presence of staphylococci characterize the morbid anatomy.

Symptoms This disease begins with one or more vegetating areas or papulopustules which soon coalesce and form plaques of exuberant granulation tissue. The areas are usually dark red in color and are covered with purulent crusts. The affected areas are elevated sharply defined and enlarge peripherally by the formation of new vegetating areas. The lesions are often malodorous. The sites of predilection are the genitalia, inner surface of the thighs, and lower abdomen. The subjective symptoms consist of itching and slight pain.

Diagnosis It is differentiated from blastomycosis by the absence of Blastomyces. The history of having taken bro-

mides is absent in dermatitis vegetans. It is distinguished from pemphigus vegetans by its promptness in responding to antiseptic treatment.

Prognosis The disease may continue for many months or years; however antiseptic treatment and cleanliness finally effect a cure.

Treatment Application of wet dress-

ings of potassium permanganate 1:5000 followed by the application of 5 per cent ammoniated mercury ointment or 5 per cent sulfathiazole ointment, usually produces a cure. Roentgen therapy often causes prompt involution and a cure. The oral administration of the sulfonamides especially sulfathiazole, is worthy of trial in resistant cases.

DERMATOMYCOSES

Dermatomycoses are cutaneous diseases produced by various kinds of fungi or Hyphomycetes.

Etiology More than 100 species of fungi are known to affect the human skin. The following are the most important ones: *Microsporon*, *Trichophyton*, *Epidermophyton*, and *Achorion*. *Microsporon*, *trichophyton*, *epidermophyton*, and *favus* are the clinical names given to the diseases caused by these fungi.

Trichophyton (ringworm) manifests itself differently in certain parts of the body presenting distinctive features characteristic of the affected part. The disease for this reason is divided into (1) *trichophyton corporis* (ringworm of the body) which includes ringworm of the hands and feet as well as *tinea circinata* and *tinea cruris*; (2) *trichophyton capitis* and *kerion*, applied to ringworm of the scalp; (3) *trichophyton barbae*, applied to ringworm of the bearded region, and (4) *onychomycosis*, which is a fungus affection of the nails.

Ids Secondary localized, or widespread eruptions of allergic character in which fungi are not present may be associated with some of the common especially severe or deep cutaneous mycoses. The allergic substances which originate from fungi are intracutaneous and are disseminated by the blood and lymph

stream. These fungotoxins produce in remote sensitized areas of the skin, eruptions analogous to the tuberculids. These secondary allergic eruptions are termed epidermophytids, trichophytids, microsporids, favids, monilids, etc. each organism forming its own id. These dermatophytids present a varied appearance and occur chiefly on the trunk and extremities and, according to Preck, are either epidermal, dermal, subcutaneous, or vascular. The eczematous variety is invariably present where the epidermis is involved. Where the dermis is involved, psoriasisform or lichenoid eruptions occur. Subcutaneous lesions simulate erythema nodosum, while vascular manifestations occur in the form of urticaria.

While a number of types of trichophytids have been described, the varieties most often seen are vesicular or eczematoid, as in dermatophytid of the hands associated with dermatophytosis of the feet. Erythema-scarlatiniforme and erythema multiforme type lesions have also been observed. In the kerion type of *tinea capitis*, a lichenoid eruption may be present in areas remote from the original site. The lesions are single or multiple, grouped or disseminated, discrete, pale pink, conical or flat papules. All ids are self limited.

A filtrate from fungus culture (referred to as trichophytin) when injected intra

cutaneously into persons infected with similar fungi will produce a cutaneous reaction not unlike a tuberculin reaction. In positive reaction an erythema or a

with the patch test method, the reaction is characterized by a papulovesicular dermatitis. It usually occurs in a few days or it may be delayed for several weeks. This method is not very reliable.

A positive trichophytin reaction denotes sensitization to fungus, while a negative trichophytin reaction proves that no sensitization to a fungus has occurred. If this should occur in the presence of a proved fungus infection, it means that the infection is very recent or that the individual's defense mechanism is poor. Before sensitization occurs, fungus must be present at least two weeks.

A negative trichophytin test in the presence of a dermatitis in which fungi are absent gives additional proof that the eruption is nonmycotic.

According to Lewis and his coworkers, the specificity of the test is chiefly determined by the genus. The reaction to trichophytin is always positive when the subject has a trichophytid. In fact "ids" may be precipitated by trichophytin injections.

Tinea Circinata

SYNONYMS: *Trichophytosis corporis*, *tinea trichophytina corporis*, *herpes circinatus*, ringworm of the body

Tinea circinata is an inflammatory contagious affection of the glabrous skin.

Varieties. Several types are described namely the macular vesicular tinea profunda (trichophytic granuloma), agminate folliculitis, and perfolliculitis suppurativa conglomerata of Leloir.

Incidence. This disease attacks both sexes and occurs more frequently during childhood and adolescence. It seldom occurs after middle life.

Etiology. The *Trichophyton* or large-spored ringworm is the cause of tinea circinata.



Fig. 202 Trichophytids.
Small and papular

papulovesicular lesion surrounded by an area of erythema occurs within forty-eight hours. When trichophytin is used

Pathology The fungus can be demonstrated by placing scales or the everted roof of a vesicle on a glass slide adding a few drops of a 10 per cent aqueous solution of potassium hydroxide, and covering it with a cover glass. Moderate heat is applied with a Bunsen burner or flame from a lighted match until the

papillary layers of the corium. The horny layer is the seat of parakeratosis. Vesicles containing serum and leukocytes can be found just beneath this layer. Depending upon the intensity of the process, varying degrees of edema and leukocyte infiltration are present in the rete malpighii.



Fig. 203 *Dermatophytids*. (Courtesy of Dr. C. C. Thomas.)

material is macerated. The slide is studied under the low power of the microscope. Fungus hyphae and spores are usually plainly seen. The fungus may be stained with polychrome methylene blue or Welch's stain. Material obtained in the same way as for microscopic study may also be cultured by planting it on Sabouraud's proof medium.

The pathological changes are limited to the epithelium, papillary and sub-

Symptoms *Tinea circinata* is characterized by one or more pea sized, nummular or larger circular circumscribed, slightly erythematous, dry scalp patches. These may not be raised above the level of the normal skin or may be slightly elevated particularly at the border. The border is usually more inflamed and more scaly than the central portion of the patch which may appear to be unaffected to the naked eye. An annular

outline is thus produced so that the ring (ringworm) may attain a diameter of several inches. In some cases, concentric circles develop making various patterns. The patch is usually from $\frac{1}{2}$ to 1 inch in diameter and after reaching



Fig. 201: *Microsporia*. Occurred in four of children in the same family as well as their cat and dog.

this size, often remains stationary or disappears.

In other varieties of *tinea circinata* there is a more marked tissue reaction to the fungus which is characterized by *vesiculation and exudation*. As this inflammation spreads peripherally circular crusted patches are formed. Like the macular type these have a tendency to clear at the center while spreading at the periphery so that ringed thickly crusted lesions are produced. These lesions as a rule cause very little discomfort other than slight itching; occasionally they spread acutely and cause a moderate constitutional reaction in children.

Multiple disseminated patches of both the dry (macular) and moist (vesicular) types of *tinea circinata* are encountered, in which case most of the surface of the skin is involved. A deep pustular type of *tinea circinata* which is follicular and has a mucopurulent or seropurulent discharge resembling carbuncle or kerion is sometimes observed. This is called *trichophytic granuloma* or *tinea profunda*. It is a circumscribed annular or circular raised crusted boggy patch, in which the follicles are distended with viscid purulent material (*perifolliculitis suppurativa conglomerata* of Lelorr).

The deep-seated variety of *tinea circinata* may flatten centrally and extend peripherally and is then called *agminate folliculitis*.



Fig. 203: *Tinea Corporis*. (Courtesy of Dr. Jacques P. Gaequiere)

Diagnosis. *Tinea circinata* must not be mistaken for *seborrheic dermatitis*, *pityriasis rosea*, *psoriasis*, *neurodermatitis*, or *lupus erythematosus*. The discovery of the fungus either by micro-



Fig. 206 Thin Crania. (Courtesy of Dr. Carroll S. Wright.)



Fig. 207: Tinea and Chemical Dermatitis. (Courtesy of Dr. Carroll S. Wright.)



Fig. 208 Ringworm (with secondary erysipelas) (Courtesy of Dr. Carroll S. Wright.)



Fig. 209: Tinea (hyperkeratotic) Not uncommon but unusually extensive form of chronic acromycosis. (Courtesy of Dr. Carroll S. Wright.)

scope examination or by culture makes the diagnosis a certainty.

Prognosis The course of *trinea circinata* varies with its different forms. The superficial form may persist indefinitely or it may spontaneously disappear. The inflammatory type runs a shorter course and often spontaneously disappears. The deep-seated types often

with alcohol or water (about 1 ml official tincture of iodine to 6 parts of diluent) *Sulfur* or *Whitfield's ointment* is equally efficacious. If the lesions are edematous and erythematous, *wet dressings* are the treatment of choice; any of the following are recommended: potassium permanganate (1:8000), liquor aluminum acetate (1:15) or resorcin (2 per cent)



Fig. 210 *Trinea Circinata*. Left Due to an animal ringworm. Note the well-defined lesions showing numerous pustules and scale-crusts. Microscopic examination was positive for ringworm. Right Of the face.

leave areas of atrophy. The prognosis is favorable.

Prophylaxis Soap and water should be used and the skin thoroughly dried followed by the use of a nonallergic dusting powder. Avoidance of contact with persons, animals, or birds affected with *trinea* is essential.

Treatment The macular and vesicular types of *trinea circinata* readily disappear following the daily application of tincture of iodine for a period of three days. In the case of children, it is well to dilute the official tincture of iodine

In the profunda and follicular types, 0.5 per cent aqueous solution of *argenti nitrate* is effective. Five per cent *salicylic acid* ointment is a very successful treatment for the suppurative type.

One per cent *gentian violet* in alcohol or water applied three times each day is also satisfactory. Fifteen to 20 per cent *salicylic acid* in alcohol or 10 to 30 per cent *resorcinol* in equal parts of alcohol and ether may also be used. When the affected part becomes dry and scaly ointment and paste are indicated. An ointment or paste containing 1 to 3

per cent *mercury salicylate* or 2 per cent *ammoniated mercury* or one half strength *Whitfield's ointment* is the usual method of treatment.

If the lesions are greatly infiltrated occlusive *hot aqueous dressings* are definitely indicated. Several thicknesses of gauze soaked in 3 per cent hot aqueous solution of resorcin covered with wax paper and applied overnight and repeated for a period of two hours several times during the day for several days is recommended.

Radiotherapy shortens the convalescence of deep trichophytosis. It is given in two doses of epilation strength ten days apart.

Tinea Cruris

SYNONYMS: *Epidermophytosis cruris*, *tinea trichophytina cruris*, *eczema m. glinatum* *Jock Itch* *red fl p dhobie itch*.

Tinea cruris is caused by the *Epidermophyton inguinale* which is a fungus closely related to the trichophyta.

Incidence It is more common in males than in females.

Pathology The fungus *Epidermophyton inguinale* forms a yellow and powdery culture and has the peculiarity that it attacks the stratum corneum but never attacks the hair.

Symptoms *Tinea cruris* occurs upon the upper and inner surfaces of the thighs, being popularly known as "jockey strap itch." In the tropics it is called dhobie (laundryman's) itch or "red flap." It begins as a small erythematous, scaly or vesicular crusted patch which spreads peripherally and partly clears in the center. The patch is characterized by its curved, well-defined border particularly on its lower edge. It may extend downward upon the thighs or backward on the perineum or about the anus. At

times the penoscrotal fold or sides of the scrotum are involved. The disease may affect both groins, but more often is largely if not entirely on one side.

Diagnosis The diagnosis depends on the clinical appearance and localization and on the demonstration of the fungus.



Fig. 211: *Tinea Cruris*.

in the scales and vesicles of the border. This form of epidermophytosis must be differentiated from erythrasma, from moniliasis of the groin from the superficial form of dermatomycosis *trichophytica*, from intertrigo from seborrheic dermatitis, and from neurodermatitis. The color of erythrasma is brownish red, the patches are not elevated, and on microscopic examination a different fungus is found. Moniliasis usually exhibits small marginal areas of pustulation and microscopic examination shows a different fungus. Dermatomycosis *trichophytica* is characterized by a more rapid course and may involve the hairs of the inguinal and pubic regions. Intertrigo lacks the pronounced and sharply

demarkated border and on microscopic examination no fungus will be found. Seborrheic dermatitis is characterized by its uniform, smooth red surface, and sharp definition and the absence of fungus on microscopic examination. The history of neurodermatitis, the presence of symmetrical lesions elsewhere on the cutaneous surface, the frequent presence of lichenification and the absence of fungi on microscopic examination differentiate this disease from *trinea cruris*.

Prognosis. The course of the disease is persistent and relapses are common. The prognosis under proper therapy is good.

Prophylaxis. Careful drying after bathing, prevention of excessive perspiration and chafing together with the use of mild antiseptic and nonallergic dusting powder are prophylactic measures which may be tried to advantage.

Treatment. Soothing applications such as a saturated solution of boric acid, calamine lotion with 1 per cent ichthylol, or 1 per cent solution of aluminum acetate, should be employed until the affected areas can tolerate parasitocidal remedies. After the inflammatory symptoms have subsided, a 10 per cent solution of sodium thiosulfate applied twice each day is of value.

The following prescriptions have been successfully used:

I	
T. of zinc	10.0
Urechol	50.0
W. ter q. oil	100.0

Use. Paint on affected areas three times each day and apply talcum powder.

II	
Bismarck	2 per cent
Zinc oxide	20.0
Talcum	20.0
Glycerin	10.0
Alcohol	50.0
W. ter	50.0

Use. Apply twice each day.

III	
Chrysarobin	0.3
Liq. collodion	30.0
Use. Apply to affected areas 1.5 cc each day	
IV	
Precipitated sulfur	1.0
Acid salicylic	1.0
Ung. aquaphor	30.0
Use. Apply twice each day	

Therapy is continued until scaling occurs. When this has been accomplished, soothing lotion such as calamine lotion, are indicated. The peeling treatment may be repeated if necessary.

Röntgen therapy given in small fractional dosage (60 to 75 r) with the acetum carefully screened with lead, may lessen the eczematous phase.

Dermatophytosis Pedis

SYNONYMS. *Athlete's foot, golf foot, dermatomycosis epidermophytosis of the feet, eczematoid ringworm of the extremities.*

Dermatophytosis of the feet, commonly known as athlete's foot, is an inflammatory contagious, cutaneous dis-



Fig. 212. Tinea Dermatitis.

ease of the feet, which may also involve the hands as a direct infection and in the form of "tids."

Varieties. This affection may appear in three types: the vesicular or dyshidrotic; the intertriginous; and the hyperkeratotic.

Incidence About 50 per cent of vesicular lesions of the feet in adults is due to fungus infection. It is less common in children. Men are more often affected than women and the affection is more prevalent in summer exacerba-



Fig. 213: Squamous Nodular Tertiary Syphilid. Of sole of foot, simulating ringworm.

tions occurring in the spring or beginning of summer.

Etiology The common etiological factor in dermatophytosis is the fungus known as the *Epidermophyton interdigitale* however several other varieties of the *Epidermophyton* genus, such as the *Epidermophyton inguinale* and the *Epidermophyton rubrum* have also been cultured from the lesions. The same clinical picture may also result from infection by the *Trichophyton* which is found in 10 per cent or more of normal feet.

Pathology The fungus or causative organism is usually found in large numbers in stained sections in the horny layer as short or long septate, refrac-

tile, homogenous or slightly granular mycelia which tend to run parallel to the surface. Exceptionally they may be found in the vesicle and in the lower layers of the squamous cells which form in the roof of the vesicles. Histologically it resembles eczema however fungi are absent.

Symptoms The dyshidrotic or vesicular variety usually begins with a deep-seated slightly elevated vesicle which contains a clear fluid. Usually there is no surrounding edema. After a few days the fluid of the vesicle is absorbed and a brownish macule marks the site of the original vesicle. Other vesicles appear on the ventral surface of the toes and under the arch of the foot and finally the entire sole may be involved. The vesicles are usually about 2 or 3 mm in diameter. They occasionally coalesce and form bullae of various sizes. They have



Fig. 214: Wartlike Trichophytic Granuloma. Outer side dorsum foot.

a bluish tint. Burning and itching are the chief subjective symptoms. These may be relieved by opening the vesicles and emptying them of a clear glycerine-like fluid.

The squamous hyperkeratotic variety is characterized by an erythematous skin



Fig. 215 Eczematoid Ringworm. Due to the Epidermophyton.

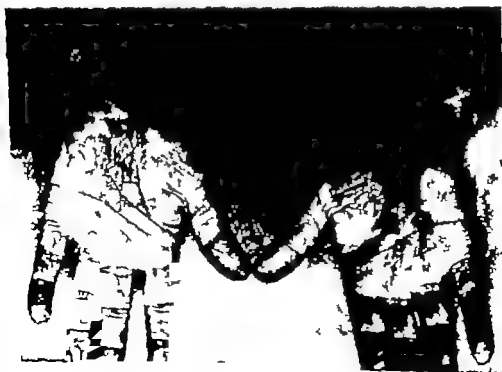


Fig. 216 Squamous Thymal Dermatitis.

which soon becomes keratotic and scaly. The sites of predilection are the soles, sides, the dorsa and the toes. Occasionally a diffuse keratoma of the entire sole results. This variety is the result of infection by the *Epidermophyton rubrum*. Fungi are readily demonstrated by microscopic examination.

the only part affected. Fungi are readily demonstrated by microscopic examination from the scraped epidermis. Hyperhidrosis is invariably present and a certain amount of maceration between the toes is evident. Scaling between the toes, to a varying degree, is a common finding on routine examinations. Wexler has



Fig. 217 Squamous Tinea Dermatitis.

The intertriginous variety is the most common. The primary lesions often consist of patches of white sodden epidermis and of fissures between and under the toes. Ofttimes, the only clinical sign is a fissure at the base of the fifth toe. One or all of the interdigital webs may be affected; however the skin between the fourth and fifth toes of each foot is often

shown that such simple scaling is not always tinea. At least it is his conclusion that fungi are absent microscopically and culturally in a definite percentage of these cases.

Diagnosis. This affection must be differentiated from dyshidrosis, contact dermatitis, and pustular bacterid. *Dyshidrosis* usually starts on the dorsal rather

than on the ventral surface of the toes and it more often starts on the great toe. Microscopic examination of the scrapings from the affected area determines the diagnosis.

Contact Dermatitis This occurs primarily or secondarily in a certain number of patients with dermatitis of the

feet. It is due to sensitiveness to various medications applied for a real phytosis or primarily to shoe polish or chemicals in leather or coverings of shoes or of slippers or dye in hose and stockings. It is seen, as a rule only on the skin of the dorsum of the foot, rarely the sole. Fungi are absent in the latter forms and

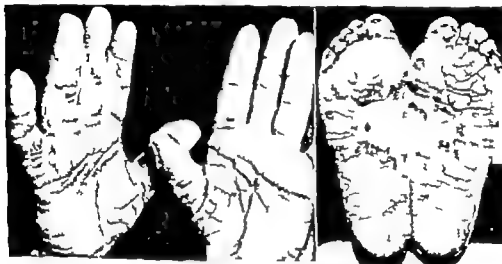


Fig. 218 Dermatomycosis (hyperkeratotic) Left Of hands. Right Of feet.



Fig. 219 Epidermophytosis. Squamous variety of fungus infection of foot.

patch tests with suspected substances may be necessary.

Pustular Bacterid (Andrews) The primary lesions are minute, grouped often hemorrhagic vesicles which rapidly develop into pustules. These appear in pruritic successive crops on localized



Fig. 220: Epidermophytosis. The lateral borders of the toes are often involved in the inflammatory form of epidermophytosis. Most objectionably symptomless types, however, are characterized by mild or moderate interdigital scaling and occasionally fissuring

areas of the soles and palms and often at points of pressure on these. The condition may be unilateral or bilateral and is extremely resistant to therapy. After repeated outbreaks, the affected areas become covered with adherent, dry psoriasisform scales, which accounts for the misleading term "pustular psoriasis," which has been given to the condition. Local treatment is of little value, cure often follows removal of a distant focus of infection such as in a tooth socket,

tonsils sinuses, etc. (see also Dermatitis Repens, p 292)

Complications The fissures between the toes, as well as the vesicles, may be secondarily infected with pyogenic cocci and then cellulitis, lymphangitis, or erysipelas may result. According to Traub, Tolman and Weisman these complications may also be "rids" or may be due to the fungi rather than to hemolytic streptococci as is usual. Eczema is also a common complication.

Prognosis Epidermophytosis of the feet is usually very obstinate; however, difficult as it may seem, a cure follows continuous rational treatment.

Prophylaxis It is necessary to avoid infection from bathroom floors, swimming pools, or dressing rooms. The skin must at all times be kept dry and hyperhidrosis, if present, must be treated. Avoidance of tight fitting shoes is an important prophylactic measure. If one is exposed to possible contamination from contacts, it is advisable to use the following dusting powder as a daily routine:

Menthol	40
Acid boric	500
Campbor	50
Acid salicylic	20
Titanium oxide	15
Talcum q.s.	100

Immersing the feet in a 1 per cent freshly prepared solution of sodium hypochlorite may prevent infection.

Epidermophytosis of the Hands Epidermophytosis of the hands is caused by the same group of fungi which are causative of a similar condition affecting the feet. The same varieties are present on the hands as on the feet. Involvement of the hands is not nearly so common as that involving the feet.

The dyshidrotic form of epidermophytosis of the hands has the same clin-

real characteristic as that of the feet. The clinical picture resembles that of dyshidrosis except that the fungus infection of the hands tends to be unilateral and the lesions are usually less disseminated. The microscopic demonstration of the fungus is the only means of differentiation.

The squamous hyperkeratotic variety also corresponds to that of the feet. The entire palm may exhibit a slight erythema associated with keratosis and scaling. Isolated patches may occur on the dorsum of the hands and fingers and

hidrosis is difficult. The two conditions may occur simultaneously; however freedom from recurrence of the vesicular eruption after elimination of the primary epidermophytic focus is conclusive evidence that one is dealing with dyshidrosis.

Peck and his collaborators give the criteria useful in the differential diagnosis of contact or industrial dermatitis on the hands from trichophytids as follows:

1. An active fungus infection of the feet must be present to make a diagnosis of a trichophytid of the hands.

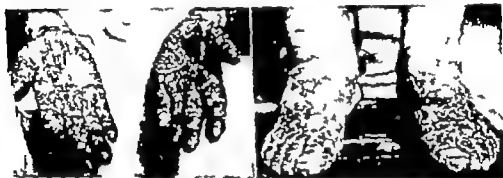


Fig. 221. Epidermophytosis.

often resemble callus. The intertriginous form of epidermophytosis of the hands is rare and when present is not dissimilar to monifial infection (*crasso interdigitalis blastomycetica*). Microscopic or cultural demonstration is necessary for diagnosis.

Dermatophytid of the feet, ankles, and callosities is of common occurrence. The lesions are most often pruritic eczematoid patches of various sizes. Less often such an eruption may involve large areas of the trunk, as well as the limbs. Lichenification due to rubbing may be superimposed upon any of these manifestations and at times may completely obscure the original condition.

Diagnosis. Differentiation from dys-

2. Trichophytin tests must be positive before a diagnosis of trichophytid can be made.

3. Trichophytids do not show the rapid improvement an industrial dermatitis will show after removal from contact with the known industrial irritant. Trichophytids do improve under suitable therapy even if patients continue their work.

4. An industrial dermatitis most often occurs on the dorsa of hands, whereas an "id" commonly appears on the palms and flexural parts of the sides of the fingers; furthermore, both hands are usually involved.

5. Negative patch tests with the suspected sensitizers tend to rule out an

industrial dermatitis. In this connection Schwartz has shown that about 80 per cent of all cases of industrial dermatitis are due to primary irritants and only about 20 per cent are due to sensitizers.



Fig. 222: Epidermophytosis. Infection of both palm. Note de-epitheliation over palmar surfaces with deep-seated crusts.

6. A positive trichophyton and a positive patch test with an active fungus infection may indicate an "id" combined with a contact dermatitis in the same person. Further observation in such cases is always necessary.

Treatment. When the condition is acute and pyogenic infection is present wet dressings and lotions constitute the treatment of choice and it is only after the acute symptoms have subsided that recourse may be had to the more active antiparasitic and fungicidal medications. The wet dressings of choice are 1 per cent aluminum acetate, a 0.25 per cent aqueous solution of silver nitrate, 3 per cent solution of boric acid, 2 per cent

solution of resorcin or potassium permanganate, 1:10,000. Calamine lotion with 2 per cent ichthyol is preferable for treatment of the subacute form. Soap and water is contraindicated in the acute forms and should only be resumed after all symptoms have subsided. The fungicides are almost numberless and cure everything except ignorance. The following fungicidal agents have proved themselves clinically effective: iodine, benzoic acid, thymol, salicylic acid, resorcin, sulfur, gentian violet, sodium hypochlorite, ammoniated mercury and formaldehyde. The fungicide most suitable for the case depends upon the location of the disease on the skin and the stage of eruption.

Salicylic acid and resorcin are fungicidal and have a keratolytic effect. They



Fig. 223: Tertiary Syphilitic (simulating tinea dermatitis). Note involvement of the ulnar surface of the wrist as well as the entire palmar area. It was unilateral and had been present eight years, during which time it had been treated as a tinea and tinea dermatitis.

are most important where the fungus is protected by a thick layer of overlying skin such as that on the soles. Salicylic acid is, however, the more widely used and probably the better of the two medicaments. The strength varies from 2 to 5 per cent, depending upon the degree

of exfoliation required. Whitfield's ointment (6 per cent salicylic acid and 12 per cent benzoic acid) is too strong for all but very tough areas of skin. For tender areas, one quarter to one half strength is preferable. Resorcin may be employed in strengths of from 5 per cent to 30 per cent.

Two measures have been found effective in making the skin less susceptible to invasion by fungi: (1) use of *dusting powders* and (2) use of *weak acids* to lower the pH of the skin. Both purposes may be accomplished by using boric acid in a dusting powder. Talcum is the usual base and fungistatic powders, such as thymol, iodine, or sulfur may be added in suitable strength. Marchionni used concentrated hydrochloric acid, 6.4 cc. in 100 cc. of 70 per cent alcohol, painted on the skin several times a day for the purpose of lowering the pH of the skin. The pathogenicity of both bacteria and fungi seems to be related to tolerance of the skin for different degrees of pH.

Excellent results have been obtained by Peck and Rosenfeld with the following:

Undecylenic acid	80
Zinc undecylenate	20.0
Greenish oil base	100.0

Sec. This cream should be managed in thoroughly at bedtime; for day use, the active ingredients may be used as powder substituting talc for the greenish base. Both may be used for prophylactic and therapeutic purposes.

The following prescriptions are suggested by George Andrews:

I

Thymol	0.12
Acid salicylic	4.00
T. iodine	24.00
Alcohol, 85 per cent q	30.00

Sec. Paint on terminals and bet. can toes once day

The above prescription should be used half strength for tender or sensitive skin. It may be alternated with a soothing preparation in case of irritation.

II

Acid benzoic	1.0
Acid salicylic	1.0
Paraffin molle	5.0
Paraffin dur	1.0
Oil cocco sativus q. s.	30.0

Sec. Whitfield's ointment. Rub into affected areas morning and night.

III

Resorcin, 5: 30 per cent in alcohol

Sec. Apply to affected areas morning and night.

IV

Acid salicylic	1.0
Boliver ppt.	5.0
Adipic lauric by d. q. a. ad	30.0

Sec. Apply to affected areas morning and night.

V

Chrysarobin ointment, 2 per cent

VI

Aq. sol. gentian violet, 1 per cent

Sec. Paint on affected areas morning and night.

At the University of Chicago the routine treatment consists of a *sulfur salicylic acid ointment* applied twice daily and painting the affected areas with 2 per cent iodine in benzol. The ointment, consisting of 1 per cent sulfur and 0.5 per cent salicylic acid, may be made more active by increasing the sulfur content to 3 per cent and the salicylic acid content to 2 per cent.

Acid salicylic	1.0
Resorcin	1.0
Acetone	1.0
Alcohol, 70 per cent q	30.0

Sec. Apply for three days only

Castellani's paint is effective in some cases.

Sa. W.'s paint is also a popular remedy.

Acid salicylic	5.0
T. mercuriodate	100.0

The following is of special value for scaling between the toes (usually tinea)

Oil cloves	0.1
Oil cinnamon	0.1
Iodine (crystals)	1.0
Potassium iodide	2.0
Acid salicylic	2.0
Acid benzoic	4.0
Sol. vini. rect. (70°)	100.0

Used regularly it will stop and prevent recurrence of the scaling. Although there are incompatibilities in this formula its activity is maintained for four to six weeks or longer.

The only physical method that is of any great value in the treatment of dermatophytosis and dermatophytid is x ray therapy in fractional dosage. Here it usually results in the same pronounced benefit seen in other eczematoid skin lesions. Nearly all clinical varieties of dermatophytosis are improved by a few fractional doses of x rays. This is particularly true of the acute vesicular eruptions of the hands and feet whether the lesions are direct infections or dermatophytids. Along with roentgen rays, wet dressings of 1:10 dilution of liquor aluminis acetatis or boric acid solution or other soothing remedies should be used until the acute stage subsides then more active fungicidal measures may be employed. Chronic intertriginous lesions, as a rule, respond more quickly if several fractional roentgen ray treatments are given. The same is true of chronic keratotic types of dermatophytid.

The dosage usually employed is 50 to 150 r units of unfiltered radiation at intervals of five days or longer. Usually three to six treatments suffice. Great care must be taken to avoid excessive roentgen ray treatment in recurrent attacks of epidermatophytosis or dermatophytid. Generally the same areas are involved repeatedly and too

many short courses of x ray treatment may result in radiodermatitis. X ray treatment is palliative but not curative. Roentgen therapy does not kill fungi. On areas invaded by fungi, topical destructive means must be used. Dermatophytid responds well to x rays and mild local applications but will usually recur unless the primary foci are eradicated.

The treatment of the lichenification which may accompany or supersede epidermatophytosis is irradiation. Generally a few fractional exposures bring about the desired result, the itching being frequently controlled by the first treatment. The dosage should always be below that which produces epilation or erythema.

Dermatopolymyositis

SYNONYMS: *Dermatomyositis*, *primary polymyositis*.

This is a rare disease characterized principally by a degenerative non-suppurative inflammation of many muscles associated with a variety of skin changes. It is often fatal. In Stern's collection of twenty-eight cases, seventeen died.

Etiology. The exact cause is unknown. Acute infections (influenza, respiratory tonsillar sinus) have preceded the development of the disease in many cases. In the series studied by O'Leary and Waisman foci of infection were present in twelve. Bacterial toxins with a special muscle tissue affinity appear to be the chief factor.

Symptoms. The onset may be acute with severe muscle pain or it may be and usually is, gradual with mild muscle pain, worse on movement or pressure, and either generalized or limited to the extremities. The involvement is often symmetrical and the muscles are not only

painful but tender. The muscle atrophy which develops later is associated with contractures and limitation of joint motion. There is weakness (especially later) firm diffuse pitting or nonpitting edema, and skin lesions in most cases. Moderate fever stomatitis, diffuse alopecia, splenomegaly, calcinosis cutis, sclerodermatous changes, and cardiac arrhythmias are not unusual. The skin lesions consist of a superficial, diffuse, or mottled erythema especially over the edematous areas, sometimes clinically but not histologically resembling those seen in disseminate lupus erythematosus. Mild urticarial lesions may occur and sharply defined, patchy pigmentary and atrophic skin changes accompany or often follow the erythema.

Diagnosis. Diffuse scleroderma is pro-

gressive but differentiation in early cases may be altogether impossible. Sclerema adultorum tends to complete involution in several months to several years. It is characterized by a firm swelling of the chest wall and neck and the skin is deeply indurated. In trichiniasis, there is often a history of familial attacks, marked eosinophilia facial (especially periorcular) edema, and a positive intradermal Bachman test. The differentiation from subacute disseminate lupus erythematosus is at times difficult and may require time. Blood alterations seen in lupus erythematosus are not present in dermatomyositis.

Treatment. It is largely symptomatic. *Rest is essential.* *Nonspecific protein therapy* may be of value. The *sulfonamides* and *penicillin* are worthy of trial.

ECTHYMA

SYNONYMS: *Ecthyma ulcerans*

Ecthyma is a pustular infection of the skin not unlike impetigo contagiosa except that it is deeper situated and is characterized by large flat crusts.

Incidence It is more common in adults and those who are improperly nourished. It is also seen in cachectic states such as cancer, anemia and tuberculosis.

sores begin as or become additionally infected ecthymatous lesions. In the persistent forms of these conditions, certain constitutional diseases, syphilis in particular must be taken into the etiological consideration.

Differential Diagnosis The disease is differentiated from impetigo contagiosa and crusted syphilides. The lesions



Fig. 224: Ecthyma Gangraenosum. Seven week duration. (Courtesy of Dr. H. L. Blair)

Etiology The causative organism is the same as that of impetigo contagiosa. The staphylococcus or streptococcus is probably causative; however, a large number of organisms have been found associated.

Symptoms Lesions occur anywhere on the integument, but the buttocks, thighs, and legs are common sites. Children are more frequently afflicted than adults. The lesion first appears as a tiny bulla resting upon an erythematous base. This is followed by the appearance of a grayish yellow or blackish brown crust. The crust is a thick mass of dried pustular exudate. Each lesion is surrounded by an inflamed, infiltrated and painful zone. The disease is infectious and is automucible. It is possible that many of the so-called veldt desert, tropical Florida and salt water

of impetigo contagiosa are more superficial and are free from ulceration. Crusted syphilides are more infiltrated, occur in the late secondary stage of lues, and other signs of lues are present.

Prognosis The prognosis is favorable in spite of the long period ranging from four to eight weeks necessary for lesions to heal.

Treatment Systemic and topical treatments are necessary. Systemic medication consists of administering general tonics in the form of iron and cod liver oil.

Local applications of a solution of 2 per cent silver nitrate or a 2 per cent solution of tincture of iodine are beneficial.

Fomentations with 1:2000 bichloride of mercury applied frequently for several days, and followed each day by painting

lemmons with a 3 per cent solution of silver nitrate in sweet spirit of niter are decidedly helpful.

Ultraviolet light administered daily in erythema doses, prevents the appearance of new lesions. Fractional unfiltered x-ray therapy of 80 r ($\frac{3}{4}$ R.D.) given once a week, ends in recovery after four or five exposures.

Ecthyma Gangraenosum

SYNONYMS *Ecthyma trochanicum*, *infection*, *dermatitis gangraenosa*, *infection*, *caricula gangraenosa*, *pyrophagus gangraenosa*.

Ecthyma gangraenosa is a rare form of ecthyma occurring in debilitated children and as a sequel to one of the pustular exanthemas, especially varicella.

Incidence It may follow an attack of varicella, varicella, rubella, and purpura. It usually occurs in marasmic female infants and young children.

Etiology The causative factor may be the *Streptococcus haemolyticus*, the *Bacillus pyocyaneus*, virulent streptococci, diphtheroids, or a mixed infection.

Symptoms Ecthyma gangraenosa is characterized by vesicobullous eruptions which soon undergo necrosis. The lesions are round or irregular pea to bean-sized gangrenous sloughs which occur on former vesicular or pustular lesions. The gangrenous areas often coalesce to form large patches. The sites of predilection are the buttocks, thighs, and trunk. The constitutional symptoms are those of toxemia and occasionally of bacteremia. The disease may last from a few weeks to several months.

Prognosis The prognosis depends on the extent of involvement of the cutaneous surface and the virulence of the organism. Most of the patients recover. Scarring usually occurs.

Treatment The treatment of ecthyma gangraenosa is similar to that of impetigo contagiosa. Ultraviolet irradiation and x-ray therapy are occasionally helpful. A paste of zinc peroxide or an ointment containing 5 per cent sulfanilamide or 5 per cent sulfathiazole is often curative.

ECTODERMAL DEFECT

SYNONYMS *Hereditary ectodermal dysplasia of the anhidrotic and hidrotic type*, *hereditary ectodermal dysplasia*.

These terms are used to designate the results of presumably defective germ plasma which manifests itself in single or multiple defects or malformations involving the epidermis and its appendages. These defects are rare. The terms ectodermal defects, ectodermal dysplasias, "hypoplasia, and aplasia are all used freely to discuss this group of unusual conditions. When the mesodermal structures are involved, one may find absence of collagen and elastic tissue bone defects (absent patella, cranial defect) familial syndactyly and polydactyly hypoplasia of the interphalangeal joints of

fingers and toes, harelip, cleft palate, hypospadias, and epispadias).

Thannhauser believes that Werner's and Rothmund's syndromes also represent the results of multiple germ plasma defects. Werner's syndrome (scleroderma with cataracts progeria of adults) tends to occur in brothers and sisters. It is characterized by carities or premature graying of the hair premature alopecia of scalp, eyebrows, and other parts. pseudoscleroderma, poikiloderma, circumscribed areas of hyperkeratosis of the soles, trophic leg ulcers, juvenile cataracts, hypogonadism, arteriosclerosis,

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Local applications of a solution of 2 per cent silver nitrate or a 2 per cent solution of tincture of iodine are beneficial.

Fomentations with 1:2000 bichloride of mercury applied frequently for several days, and followed each day by painting

ECZEMA

SYNONYMS *Tetter* *soit* *leuon*, *dermatitis* / *unknown origin*.

Eczema is an acute, subacute, or chronic inflammatory and itching disease of the skin characterized by erythema, papules, vesicles, or pustules, or a combination of two or more of these lesions.

From the view of the pathologist, all cases of eczema are dermatitis. In the clinic the words eczema and dermatitis are used as synonymous terms. "Eczema" is the name given to an inflammatory disease of the skin which has definite characteristics and follows a definite course, the cause of which is unknown, while the word dermatitis is employed for an eruption of a similar nature, the cause of which is known to the clinician.

Varieties Morphologically eczema has been divided into two general types: a primary type, characterized by the presence of erythematous, papular vesicular or pustular lesions; and a secondary or consecutive type in which the lesions evolve from those of the primary or elementary variety. In many instances, the lesions are multiform in character from the beginning. Probably the most frequent combination is the papulovesicular. Lesions of the four primary types may be present almost from the very onset of the disease.

Primary Eczema

Erythematous Eczema This is characterized by dry, pink, or red ill-defined lesions accompanied by some itching and burning. Involved areas may be slightly swollen and edematous. Swelling and edema are seldom noticeable unless the lax tissues of the infraorbital and scrotal regions are involved. The patches tend to spread and coalesce. They exhibit within a few days branny scaling asso-

ciated with dryness and roughness of the skin. The course is chronic with slight remissions and relapses. The primary lesions of erythematous eczema are large, irregular ill-defined erythematous patches which may involve the entire body surface. Erythematous eczema resembles in some respects the acute exanthemata.

Papular Eczema This form is characterized by numerous discrete lesions consisting of papules, varying in size from a pinpoint to a pinhead and in color from violaceous to pink. Some papules develop minute apical vesicles which ooze on excoriation. Many of the papules are capped with crusts. The lesions are occasionally follicular in character. The patches are symmetric and vary in size as well as in degree of inflammation and infiltration. The trunk and limbs are the sites of predilection although no region is exempt from the disease.

Follicular Eczema (Eczema Folliculorum) This condition was first described as a special form of eczema by Malcolm Morris and subsequently by Unna. It is characterized by a nonsuppurative inflammation of the hair follicle, in which each inflamed follicle, in the acute stage, stands out as a red papule. These follicular papules are aggregated in small or large plaques. In some, the intervening skin is involved, in which case the red patches are dotted with elevated, unflamed follicles. Extension of the inflammation appears to be from follicle to follicle. With subsidence of inflammation or with peripheral extension of the plaque, central healing occurs, followed by desquamation and yellowish staining. Itching varies but may be intense. The lesions are located on

osteoporosis, shortness of stature, and presenility. There are abortive forms. It develops in the second decade of life. In progeria of children with nanism (Hutchinson Gilford disease) the skin is atrophic and taut on the extremities, there is lack of hair as in the progeria of adults but there are no cataracts or trophic ulcers. There is no familial occurrence of the adolescent progeria and it appears in first year or second of life. *Rothmund's syndrome* differs from Werner's syndrome chiefly in the skin changes which consist of a netlike telangiectasis, scaling and atrophic spots resembling those seen in poikiloderma atrophicum vasculare. The skin is thin and pliable and shows dull red mildly indurated zones and between these small pale whitish areas.

Two general types of ectodermal dysplasias may be considered.

Minor Ectodermal Defects This group presents various grades of congenital aplasia of the teeth, epidermis, and its appendages alone or in combination with such conditions as (1) localized or generalized atrichosis (in the hypotrichosis of arrested development the hair follicles are absent in the affected parts) (2) deformed nails or rarely onychia (3) deformation of teeth or rarely complete absence of teeth (4) complete absence at birth of areas of the epiderm and appendages, and (5) acatosis.

Major Ectodermal Defects This group presents congenital anomalies varying from deformation to complete absence of all or almost all of the cutaneous structures originating from the epiderm.

The condition is familial, ordinarily limited to the male and although its cause is not known, it is generally thought to be a rare sex linked recessive

dermatosis (i. e. transmitted by females and appearing in some of the male members) although it has been occasionally observed in the female (Goekerman). It is possible that the glands of internal secretion and the thyroid in particular play a rôle in the causation. According to Cole in localized aplasia, amniotic adhesions may be an important factor.

The classic case of major ectodermal defect is characterized by:

1 A skin which grossly is dry, white, smooth, thin, pliable and glossy with or without papular lesions on the face resembling milia or naevi.

2 A facies suggestive of hereditasphilia (i. e. a saddle nose or depressed nasal bridge with or without crenae, protuberant orbital ridges, and often thick protrusive lips).

3 Almost total absence of sebaceous glands.

4 Hypotrichosis with absence of lanugo hair from most of the body surface.

5 Dental aplasia or dysplasia.

6 Absence of sweat glands with resultant disturbance in the heat regulating function. The skin cannot eliminate the necessary amount of water to keep the temperature constant under varying external conditions. These patients are unable to tolerate heat, hot food, hot drink, or much physical exercise.

7 Absent or impaired gustatory and olfactory senses.

The actual clinical triad of ectodermal dysplasia of the anhidrotic type comprises anhidrosis, hypotrichosis, and anodontia or marked dental aplasia. In the hidrotic type, the ability to perspire is maintained in part.

Treatment Treatment is of no avail, although the patient learns that he is more comfortable when he avoids exercise, heat, and hot drinks.

Etiology The etiology of eczema is obscure or entirely unknown. The disease is not infectious and has no respect for sex, age, and environment. Heredity is not a direct factor although a predisposition may be inherited. Individuals with thin, dry xerodermatous skin are particularly susceptible.

Continued and transitory irritation from chemical, mechanical, actinic, and thermal agents produces a dermatitis which terminates in eczema in susceptible persons. Staphylococci, streptococci, and other bacteria play important roles in the evolution of lesions.

Bullcley and Dyer believe that mental strain and reflex irritation are important factors in the etiology. The rash of infants arising in connection with teething may be of reflex origin, or it may perhaps develop as a result of some dietetic error and anaphylaxis. Focal infection about teeth and tonsils is often the offending agent in adults.

Faulty diet, sensitization to articles of food and foreign proteins, nephritis, and thyroid disease have been mentioned by MacKee among the possible causes of eczema.

Pathology The first pathological sign in eczema is a dilatation of the capillaries, resulting in an erythema. This is followed by exudation of round cells about the vessels. The epidermis and upper layers of the corium then become edematous, the edema in the epidermis being both inter and intracellular a condition known as spongiosis. The intercellular edema puts a strain on the intercellular fibrils of the prickle-cell layer and some of these rupture. The cells are then pushed apart and a microscopic vesicle results. Such vesicles increase in size and are pushed up to the surface by the growth of the epidermis. These macroscopic vesicles run together

until they become of a size visible to the unaided eye. They can then be seen closely set all over the surface of a patch of eczema at a certain stage of its evolution—vesicular eczema. The horny layer over the closely set vesicles is rubbed off, and a weeping surface is left which oozes serum—weeping eczema. After a time the serum oozes less rapidly and dries into crusts—crusted eczema, or the surface may become infected with streptococci and be converted into impetiginized eczema. Eventually the horny layer reforms under the crusts, but owing to the edema of the prickle-cell layer the horn cells are not normal and parakeratosis results—scaly eczema.

An acute attack of eczema such as that described may after several relapses proceed to complete cure. In many cases, however having reached the scaly stage, it passes into a chronic state characterized by scaling, thickening, pigmentation of the skin with exaggeration of the normal ridges and furrows—hebenified eczema. Eczema may however be dry and scaly from the start and may never pass through the weeping stage. Many cases of chronic eczema are of this type.

In the dry scaly and hebenified types of eczema, there is pronounced thickening of the horny layer and of the prickle-cell layer with exaggeration of the inter papillary processes (acanthosis) and a dense cellular infiltration in the corium consisting of small round cells, polymorphs, and connective tissue cells around widely dilated vessels.

Diagnosis The characteristic features of this dermatosis are sufficient to prevent errors in diagnosis. The diagnostic features are (1) itching; (2) inflammation, (3) usually a predominance of one type of lesion, (4) the absence of a definite line of demarcation between

the extremities—particularly the extensor surfaces—and on the trunk. The condition is obstinate and tends to recur. It is probably of bacterial origin.

Vesicular Eczema This is the usual and typical form of eczema. This variety is characterized by discrete erythematous patches covered with small rounded or acuminate and thin walled vesicles. The majority of the vesicles rupture spontaneously or as the result of scratching and the patch becomes covered with crusts which are interspersed with areas of denuded epidermis, the process becoming chronic. Vesicular eczema often ends in chronic eczema rubrum. Itching and burning are persistent features.

Pustular Eczema This form is characterized by pustules which are very superficial and extend only a short distance into the follicle. In the majority of cases eczema pustulosa is the result of infection of one of the various forms of eczema by pyogenic organisms.

Herpetoid (Nummular) Eczema This is a form of vesicular dermatitis which attacks the dorsal surfaces of the hands and other parts of the extremities. It appears as round and oval patches 1 to 5 cm in diameter consisting of grouped vesicles or vesicopapules. The itchy eruption (efflorescence) gradually recedes and heals. It is apt to recur several times yearly in about the same location. It has been suggested that some nummular eczemas are atypical forms of dermatitis herpetiformis. The condition is recalcitrant to local medication although x ray therapy will lead to temporary relief. The best results follow removal of foci of infection about tonsils, teeth, nasal chambers and genitalia.

Secondary Eczema

Secondary eczema evolves from one of several forms of primary eczema. The

types of secondary or consecutive eczema include eczema rubrum, eczema madidans, eczema crustosum, squamous eczema, eczema sclerosum, eczema fissum and furrowed eczema.

Eczema Rubrum This is perhaps the commonest form of secondary eczema. It is characterized by a red and glazed surface, infiltration and swelling. It follows the elementary varieties and more especially the vesicular type.

Eczema Madidans This is characterized by syrupy sticky exudate oozing from papules or vesicles which may or may not have coalesced. When the serum dries, yellowish crusts form, hence the name "eczema crustosum."

Eczema Fissum This is a secondary type of eczema characterized by more or less deep painful fissuring of the skin. The sites of predilection for this variety of eczema are the tips of the fingers and the heels.

Squamous Eczema This is a chronic secondary type of eczema which is characterized by single or multiple, ill-defined or occasionally sharply circumscribed infiltrated patches which are covered with thin dry scales. The sites of predilection are the palms, soles, legs and scalp.

Eczema Sclerosum This is characterized by rough horny papillary and hypertrophic patches. The inflammation is very slight. Impairment of joint motility may occur if the skin over the joint should be involved.

Furrowed Eczema This is characterized by a slightly erythematous harsh and dry epidermis in which tiny linear cracks may occur. This type of eczema is rare.

Eczema in General

Incidence Eczema comprises from 30 to 35 per cent of skin diseases.

Herpes zoster is identified by its insidious onset, peculiar distribution over the course of a nerve and the associated neuralgic pains.

Psoriasis has dry scaly sharply defined lesions. It has predilection for extensor surfaces and exhibits typical bleeding points on removal of scales.

Impetigo contagiosa is characterized by small or large thin-walled vesicles which become pustular and break down readily to form yellow crusts. The involved areas are hyperemic. Impetigo of the scalp is confined to the occiput and is often associated with pediculosis capitis.

Milium exhibits discrete lesions free from itching and accompanied by considerable sweating.

The lesions of *papular urticaria* are seldom, if ever grouped. Typical urticarial wheals are invariably present in this condition and are characterized by their transience.

Pruritus is often an allergic dermatitis without characteristic lesions. Local pruritus may be due to fungus infection.

Seborrhea is characterized by abundant flow of sebum and the presence of oily yellow scales.

Prognosis. Eczema tend to chronicity rather than to an acute course. The disease is likely to relapse or recur and immunity from future attacks seldom ensues. Practically all varieties of eczema are amenable to treatment.

Prophylaxis. Prophylaxis constitutes the treatment of constitutional diseases that may be present, the correction of affection of the nervous system, the elimination of local infection and endocrine disturbances and the maintenance of a well balanced diet.

Treatment. *Internal Treatment.* The patient's general health should receive attention. The elimination of sodium

chloride from the diet has resulted in complete disappearance of cutaneous lesions within forty-eight hours.

Colchicum, *sodium salicylate* and the *salicyls* are indicated in gouty and rheumatic individuals. Pursey found the administration of *pilocarpine* useful in xerodermatous patients, and states that it ameliorates the itching. Careful search is made for foci of infection about the teeth, tonsils sinuses and prostate. Injections of foreign proteins like typhoid and colon vaccine merit a trial.

Regulation of the diet is essential in children and infants. Sandels commends a diet containing milk acidified with hydrochloric acid.

Fishes rich cod-liver oil and nourishing foods are indicated in debilitated anemic patients.

Local Treatment. Ultimate recovery depends largely upon local treatment. Soap and water are harmful. Scales and crusts are readily removed by olive oil or boric acid lotion.

The choice of local medication depends somewhat upon the location of lesions. Application of oils produces furunculosis about the axilla and scrotum if applied for longer periods than a few hours. Calamine lotion cakes when applied to hairy areas and should therefore, not be used in these areas.

Lotions are preferable to ointments in early acutely inflamed moist eczema. Aqueous solutions of 1 per cent *aluminum acetate* or 0.25 per cent *silver nitrate* serve admirably. J. C. White commends *lotio nigra* in full strength or diluted with an equal amount of lime water followed by *zinc ointment*. The lotion is dabbed on freely for fifteen minutes, allowed to evaporate and is followed by the application of ointment directly to the skin or by spreading it on cloth and then applying it. Weak

the diseased and the normal skin and (5) the chronicity and tendency for recurrence.

Differential Diagnosis. Eczema must be differentiated from erythema intertrigo, erythema multiforme, scabies, dermatomycosis, trichophytids, pityriasis rosea, sycosis, erysipelas, lichen planus, syphilis, herpes zoster, psoriasis, impetigo, contagiosa miliaris, pruritus and seborrhea.

Erythema is characterized by active congestion and is completely free from scaling and itching.

Intertrigo is characterized by red non-infiltrated areas. Itching is less than in lesions of dermatitis venenata and eczema.

Erythema multiforme presents symmetric sharply defined multiform lesions. Itching is seldom present.

Scabies is often complicated by eczema resulting from overmedication in long standing cases. The lesions of scabies are discrete (excoriated) blood-crusted papules or pustules. The sites of predilection for scabies are the dorsal surfaces of the interphalangeal webs, the anterior axillary folds, the flexor surfaces of the wrists, the penis, the flexor surfaces of hands and feet, the buttocks, and the palms and soles of infants. A history of infection of the patient's associates is corroborative evidence.

Dermatomycosis is caused by a vegetable fungus (tinea). The lesions are sharply circumscribed. Tinea of the scalp is accompanied by patchy hair loss. Microscopic examination of infected areas will reveal the offending tinea. The characteristic tinea lesions of the glabrous skin are circinate areas with slightly scaly centers and vesicular advancing margins. Differentiation may be more difficult in the axillary and genitocrural regions. The sharply defined out-

line of ringworm lesions, the presence of satellite patches, microscopic and cultural identification of the causative organism are pathognomonic.

The lesions of *tinca barbae* are circumscribed nodular and inflamed. The involved hair can be readily and painlessly extracted. The offending fungus is demonstrable by microscopic and cultural examination. Parasitic involvement of the nail produces a thickened nail plate which is friable, lusterless, opaque, and yellow. *Trichophytids epidermophytids monilids* and other dermatophytids are identified by the presence of active foci of infection somewhere on the body surface.

Pityriasis rosea is characterized by an acute onset — "herald spot, symmetrical noninflammatory lesions manifesting slight itching and its cyclic course.

Sycosis is follicular and confined to bearded areas. Extraction of the hair reveals an inflamed and swollen root sheath. Areas of involvement are moist and gummy. Numerous lesions are pustular. Vasculation does not occur in and between follicular openings.

Erysipelas is accompanied with fever and constitutional symptoms. It begins with a definite site of infection and spreads gradually. The patch is infiltrated, smooth, glossy and possess a sharply defined border. Bullae are occasionally present. There is rarely any itching in erysipelas.

Lichen planus is recognized by angular papules which coalesce to form violaceous-colored patches. Healing leaves blue or brown spots.

Secondary syphilis is characterized by a polymorphous eruption accompanied by glandular adenopathy, mucous membrane involvement, a history of a chancre and a positive Wassermann test are pathognomonic.

Eczema of the Face in Adults Eczema of the forehead may arise from hatband irritation. Eczema may arise about the perioral region from lipstick and the irritation of dentifrice, prosthetic materials, and the mercury of amalgam fillings. Eczema of the eyelids may be satisfactorily treated with calamine lotion or 50 per cent lotio nigra alternated with zinc oxide ointment. Occasional applications of 5 to 10 per cent aqueous solution of silver nitrate hastens recovery of eczema rubrum. Small doses of x rays are beneficial.

Eczema of the Bearded Area Papular vesicular and mixed types of eczema frequently occur in the bearded region. This disease in this area is not confined to the hairy parts alone but includes the glabrous skin as well. Early stages of eczema of the bearded area are treated by applications of an aqueous solution of 2 per cent aluminum acetate during the day and zinc oxide ointment or a mill tar ointment at night.

Eczema of the Breasts Eczema of the nipples is common in nursing mothers. It is often resistant to treatment. Applications of calamine ointment protect the surface with a thin oily coating and should be used in early stages of the disease. The involved areas are cleansed each day with cold cream or olive oil. fissures are painted with compound tincture of benzoin or with aqueous or ethereal solution of 2 to 5 per cent silver nitrate. After nursing, the nipples are dried with a soft towel, and a soothing, antiseptic ointment like 2 per cent boric acid or 1 to 5 per cent ammoniated mercury in equal parts of cold cream and aquaphor is then applied.

Eczema of the Hands Small groups of papules or papulovesicles resting upon reddened inflamed bases occur on the dorsal aspect of hands and fingers. The

volar surface reveals fissured and squamous lesions. Treatment consists of eliminating all sources of irritation. Water and soap are especially harmful. Cold cream and mineral oil are employed for cleansing purposes. Calamine lotion carbolized zinc oil weak tar ointment solutions of 2 to 10 per cent silver nitrate gentian violet, and x rays are of definite value. Palmar eczema is accompanied by thickening and infiltration. This is treated by strong keratolytic and reducing agents. Application of ointment, containing 10 to 25 per cent salicylic acid and 2 to 10 per cent oil of cade or 10 to 20 per cent ammoniated mercury may prove useful. Eczema of the hands may be prevented by the use of purified tallow and aquaphor diachylon ointment or cold cream. Tragecanth cream or a mixture consisting of one part rose water one part camphor water and four parts glycerin may be applied once or twice each day.

The following formula has proved most satisfactory in the treatment of fissured eczema of the palms and soles

Wool fat	40
Soft paraffin	510
Stylin 22	20
Spectromerol	30
Mineral oil	20.5
Borax	0.5
W ter	10

Eczema of the Nails Eczema of the nails is often associated with eczema of the hands and feet. The adjacent skin becomes red, swollen, dried, and fissured. Eczema of the nails is differentiated from onychomycosis and psoriasis. Hot water, strong soaps, and irritant cosmetics are avoided. X ray therapy is the treatment of choice.

Eczema of the Feet Eczema of the toes is the result of mycotic infection. The presence of hyperhidrosis aggravates

solutions of *potassium and zinc permanganate* (1:5000) are also useful.

Itching is relieved by applying solutions of 0.5 to 1 per cent *phenol* or 0.25 per cent *menthol*. Tar in the form of *liquor carbonis detergens* in strengths varying from 1 to 2 per cent may also be employed as an antipruritic. Applications of *salves*, alternated with lotions, give the best results in dry eczema. *Lassar's paste* without *salicylic acid* as in the following preparation is of definite value.

Zinc oxide	40-80
Starch	40-80
Petrolatum	300
Sig. Apply thickly as a protective and soothing paste.	

C. J. White lauds the use of 5 per cent *crude coal tar* in *Lassar's paste* in the treatment of chronic eczema.

The skin is readily cleansed from dirt, oiliness, and scales by employing *Pusey's liniment*.

<i>Pusey's liniment</i>	
Powdered tragacanth	40
Phenol	40
Glyceri	0.6
Olive oil	120.0
Water q. s. d.	400.0
Oil bergamot to perfume.	

The addition of carbolic acid or menthol to *Pusey's liniment* relieves itching.

In the absence of infiltration and in cases of only slight infiltration, a mild antipruritic powder such as *Anderson's antipruritic powder* is of definite value.

<i>Anderson's antipruritic powder</i>	
Campbor powdered	40
Zinc oxide	200
Starch	600
Sig. Dust freely several times each day.	

When *salves* are used, the dressings are changed three or four times each day.

Oily mixtures are usually more soothing and comforting than lotions and

powders. The following liniment is usually nonirritating and relieves itching.

Phenol	2.5
Milk of magnesia	100.0
Mineral oil	60.0
Pulv. borax	10
Aqueae camphor q. s.	250.0

Sig. Shake well and apply freely.

Applications possessing more stimulating action are indicated in infiltrated, advanced and subacute eczema. A 5 per cent aqueous solution of *silver nitrate* may be applied to affected areas at intervals of from two to five days. Soothing applications like *calamine lotion* are freely applied. *Vegetable or mineral tar* is the most efficient and reliable reducing agent. It is applied in ointment form in subacute, chronic, and dry eczema and in lotion form whenever moisture is present. The following ointments are useful.

I	
Solution of coal tar	50
Ung. aquaphor	300
Zinc oxide ointment q. s. ad	200

II	
Crude coal tar or flantrol	10
Ung. acid boric	80
Zinc oxide	20
Petrolatum q. s. ad	300

Sig. Apply twice each day.

Keratolytic ointments containing from 10 to 25 per cent *salicylic acid* are beneficial in circumscribed patches of eczema undergoing marked thickening.

Unfiltered *x-ray therapy* for acute inflammation is of value when given in small doses of $\frac{1}{10}$ (7 r) skin unit.

Mitchell obtained effective results with *ammoniated mercury* in eczema of streptococcal origin. Five per cent *sulfathiazole* in greaselike cream is worthy of a trial in impetiginized eczema. *Alcoholic solution of iodine* (1:1000) has definite value in this type of eczema.

the use of irritating antiseptics. Scrotal eczema is often the result of placing matches and coins in trouser pockets. A scrotal apron is worn to prevent the moist, inflamed parts from abrasion by friction from contacting the thighs. In

oranges, chocolate, potatoes, tomatoes, spinach, and milk, and at times their elimination is curative or followed by considerable improvement. When eliminating a suspected food it is well to acquaint oneself with a list of foods containing such allergens. The following, for example, is a list of foods containing eggs.

(There may be sensitivity to egg, egg white or egg yolk (chicken but not duck or pigeon eggs).)

1) Cake cookies, pies, certain breads, some brands of waffles, macaroni and spaghetti, ice cream and fudge.

2) Patent food preparations such as Ovaltine and Cornmeal.

3) (Rare) baking powders, such as Calumet Baking Powder.

(A) Eggs are used commercially in the fur industry photography and the textile industries.
 (B) The breast-fed infant, control mother diet.)



Fig. 226 Atopic Eczema. True infantile eczema or eczematoid of Darier (Courtesy of Dr. Jacques P. Guéquierre)



Fig. 227 Atopic Eczema. True infantile eczema.

anal eczema, starch is made for fissures, hemorrhoids, and dermatomycoses of the crotch, feet, and vulva. In acute eczema of these areas, mild astringent and soothing lotions are employed. An aqueous solution of 5 per cent gentian violet is an excellent antiseptic and should be used in this area. For treatment a 5 per cent aqueous solution of resorcin or aqueous solution of 0.5 per cent silver nitrate is valuable for treating thickened and infiltrated anal eczema.

Infantile Eczema. Infantile eczema is of unknown etiology and occurs during the first two years of life. Some clinicians attribute it to sensitization to articles of food, such as oats, wheat, eggs,

Inhaled allergens have also been incriminated at times, such as food odors, dust from feathers, silk, cotton and even human dander.

However most patients with infantile eczema reveal no food sensitization. Some

the eczema. Daily applications of a 2 to 10 per cent aqueous solution of *gentian violet* is worthy of a trial. *Ruggle's mixture* which consists of 2 per cent salicylic acid and 10 per cent tannic acid in alcohol is recommended particularly in chronic infiltrated and fissured cases. The toes are separated by pledgets of



Fig. 225 Varicose Eczema.

cotton or wool and *Anderson's antipruritic powder* applied. Hyperidrosis is treated by *roentgen ray therapy*.

Eczema Varicose Dermatitidis and Ulcers of the Legs (see also *Varicose Veins*, p. 815). Eczema of the legs is a common and distressing ailment of elderly persons and individuals suffering from varicose veins and venous stasis. The choice of local applications is

largely dependent upon symptoms. If marked inflammation is present, the parts are kept elevated. Oozing is controlled by applying *wet dressings* radi-
ant heat and nonirritating antiseptics, like 5 per cent *gentian violet* or an aqueous solution of *bichloride of mercury* (1:5000). The involved surface may be covered at first with *salve sands* or similar dressings, and *elastic stockings* should be worn for comfort and support. Dressings are changed twice each day. Soft and hard, soluble, *gelatin flexible dressings* as suggested by Pick, Unna, and others are particularly valuable in treating oozing and ulcerative stasis dermatitis. The soft jelly consists of fifteen parts zinc oxide, twenty five parts glycerin, and twenty five parts water. The hard jelly consists of ten parts zinc oxide, thirty parts gelatin, thirty parts glycerin, and thirty parts water. Carbolic acid (0.5 to 1 per cent), ichthyol (1 to 5 per cent) or tar (1 to 5 per cent) may be added with advantage. *Pusey's favorite mixture* consists of

Zinc oxide or powdered calamine	15.0
Celastin	25.0
Glycerol	25.0
Water	25.0

Use. Mixture is heated in water bath, then applied when in fluid state to involved areas by brush. The best plan is to paint lesions with a thin coat, then apply a layer of gauze or cotton wool and follow by one or more coatings of the gelatin mixture.

Sickle-cell anemia predisposes to leg ulcers in Negroes, more rarely in the white race. The lesions are chronic, often bilateral, simulate luetic lesions, and are not responsive to treatment.

Eczema of the Genitals and Anus. Eczema in genital and anal regions is often obstinate and rebellious to treatment. The urine should be examined for sugar. Women are cautioned about

and enveloping the head with a linen cap or stocking will prevent rubbing the head against the pillow. The affected parts are cleansed with lime water olive oil, mineral oil, or almond oil.

The infant is placed on a diet free from eggs, milk, or wheat since they are common offending allergens. A porridge of soy bean or goat's milk may be substituted for cow's milk. Rice, barley or corn should replace the wheat. There is no known satisfactory dietary treatment for allergic infantile eczema. In the majority of cases, the family history gives a valuable lead in determining the offending allergen.

In other instances, the Howe elimination diet is occasionally helpful.

HOWE ELIMINATION DIET

Diet No. 1	Diet 2	Diet No. 3	Diet No. 4
Milk, only for seven to ten days	Rice Ry. bread Beef Tomatoes Banana Grapefruit Pear Peaches Sugar Salt	Rice Z. cereal Lamb Lettuce Spinach Carrots Pineapple Apricots Pineapp Syrup Sugar	Chicken Bacon Corn Tapioca Squash Asparagus Pean Lemon Pears Peaches

Local therapy varies with the stage of eruption. Wet dressings of 0.25 per cent aqueous solution of silver nitrate, milk containing 1 per cent liquor plumbi subacetici or a 1:8000 solution of potassium permanganate are useful applications if exudation is present. The following lotion is useful when the eruption is less acute.

Liq. plumbi subacetici dilute	3.0
Ichthyol	2.0
Lote calamine $\times \text{F}$ q	250.0

White's crude coal tar ointment is definitely helpful in the chronic case. Complete exposure of the body to ultraviolet light is commendable for every case of infantile eczema. The liberal use of olive oil on the skin will prevent it from drying following ultraviolet light therapy. Roentgenotherapy is contra-indicated in infantile eczema.

Atopic Eczema. Orea, Sulzberger and others consider atopic dermatitis as the early manifestation of infantile eczema. The eruption of atopic eczema closely resembles that of lichen simplex chronicus (neurodermatitis disseminata, or Besnier's prurigo). The skin is itchy and it, as well as the produced lesions, is very resistant to treatment. The lesions are characterized by symmetrically arranged patches of chronic inflammation manifesting thickening of the dermis, scaling, exaggeration of the minute folds of the skin, and a certain amount of pigmentation. The surface is generally dry although it may be considerably excoriated from scratching. The sites of predilection, in their order of frequency of involvement, are the antecubital and popliteal areas, the sides and back of the neck, the face, head, axillae, shoulders, and thorax. The lower part of the trunk and the lower extremities are usually free from lesions (see also *Habitus Pruriticus* p. 632).

Change of climate is recommended. Rest, sedatives, autohemotherapy and injections of arsenic are helpful. Wool and silk clothing is avoided.

The following ointment has given excellent results.

Vioform (Cohn)	1.5 to 2.5
Aquaphor	90.0
Petrolat. q. s.	00.0
Ben. Apply twice daily cleanse water	

clinicians consider overfeeding indigestion and constipation as the most likely etiology.

Bamber is of the opinion that over 60 per cent of babies with eczema are greedy feeders and that 30 per cent



Fig. 228: Seborrheic Eczema. In infants, this often masks atopic eczema; it is not due to food sensitization and clears rapidly under medication and local seborrheic dermatitis. (Courtesy of Dr. Jacques I. Guequiere.)

are victims of indigestion or constipation. A familial tendency to allergic diseases like eczema and asthma is often elicited. Infantile eczema in nurslings has been observed to disappear when certain articles in the mother's diet were removed.

The sites of predilection for infantile eczema are the cheeks, forehead, chin, ears, scalp, extensor surfaces of forearms, arms, legs, and thighs. The involved cutaneous surface is erythematous and the site of closely arranged vesicles emits serum and becomes crusted on drying. The itching is often severe.

Infantile eczema is not to be confused with seborrheic dermatitis. Seborrheic dermatitis begins on the scalp as oily scales of yellow color on an erythematous base located upon erythematous areas and frequently extending behind the ears and over the face. It tends to occur about the central facial area in the vicinity of the mouth and nose.

Contact dermatitis must be differentiated from true (endogenous) infantile eczema. Among the external agents are soiled diapers, clothing (worn by baby or by the parent or nurse), wools, silk, furs, feather pillows, starch in sheets, shellac dyes on coverings of furniture, toys, dolls, orris root in face and baby powders and paints on high chairs.



Fig. 229: Allergic Dermatitis, X-ray identification (also see Habitus Pruriticus, p. 632).

In treating infantile eczema, one of the first considerations is the protection of the affected parts from scratching. Pasteboard splints over the elbows prevent the infant from reaching the face.

these, the derm remains red, then atrophies, and becomes thin. Milia or epidermal cysts are common in the thinned areas. In the final stages, the only evidences of the disease are these atropho-cicatricial areas.

Prognosis The tendency persists indefinitely. In some, it decreases with age.

Treatment All trauma must be studiously avoided and affected parts protected. Large doses of vitamin D 50,000 units daily may be of benefit.

Cutis Hyperelastica

SYNONYMS *Elastic skin, India-rubber skin, cutis elastica.*

This affection is characterized by excessive elasticity of the skin, and is present in the circus freaks called India rubber men or elastic-skin men.

Symptoms The condition is usually inherited and familial but may be acquired. The skin is apparently thinner than normal, loosely attached to underlying tissues, and remarkably elastic and supple. The amount of stretching is at times extraordinary. One can draw a skin fold out to a varying degree; released, it snaps back like a rubber band to its original appearance. The elasticity may be general or regional, and certain regions may have more elasticity than others. The elbows, knees, lips, and neck are especially susceptible.

Ehlers-Danlos Syndrome Elastic skin is one of the signs of a syndrome described by Ehlers-Danlos. The phenomena in this syndrome are (1) elastic skin (2) hyperextensibility of the joints; (3) fragility of skin and its blood vessels, with scars as a result of slight injuries; the skin breaks and splits following injuries normally harmless; (4) pseudotumors also due to trauma and of

a shiny bluish red color often caused by hematomas. Incomplete types of this syndrome are common.

Treatment Protection against trauma is important in those with skin fragility and is the only measure available.



Fig. 231 Ehlers-Danlos Syndrome. Showing hyperelasticity of finger joints (left thumb is stretched almost to the wrist), hyperelastic skin, pseudotumors over knees, and scars resulting from cutaneous fragility. (Courtesy of Dr Meyer Modlesman.)

Dermatolysis

SYNONYMS *Cutis pendula, lax skin, cutis lara, pachydermatocela.*

Symptoms The skin and subcutaneous tissue are hypertrophic, loosely attached, and elongated, so that the skin hangs in folds. The term has been applied to two different conditions. In one, hanging cutaneous folds are associated

ELASTIC FIBERS, DISORDERS OF

There are several cutaneous affections in which the elastic fibers appear to bear the brunt of the disturbance. These are epidermolysis bullosa, cutis hyperplastica, dermatolysis, and cutis verticis gyrata. Pseudoxanthoma elasticum in which the elastic tissue is fundamentally concerned is discussed elsewhere (see p. 842).

served in association with porphyria. In such eruptions, the exposed surfaces were exposed to sunlight.

Pathology. Histologically there is a deficiency or absence of elastic fibers of the skin (Engman and Mook). The injury causes a separation of the superficial layers from the deep epidermis.

Symptoms. Bullae appear within a



Fig. 230: Epidermolysis Bullosa.

Epidermolysis Bullosa

SYNONYMS. Simple trauma, the hereditary pemphigus, acantholysis bullosa, hereditary tendency to formation of bullae.

This is a cutaneous dystrophy, sometimes occurring on the mucous membrane, characterized by the development of bullae under the influence of mechanical trauma.

Etiology. It is hereditary, often familial and usually becomes manifest shortly after birth. The manifestations, however, may not appear until long after birth. Parental consanguinity at times appears to be a factor. Bullous eruptions simulating this disease have been ob-

few minutes to an hour following such mechanical injury as rubbing, walking, pressure or a blow. The bullae are usually painless; they may be flaccid or tense and may vary in size; they contain clear, sometimes bloodstained, fluid. If uninfected they heal rapidly, drying in several days as the serum is slowly absorbed. New skin forms, and may be slightly pigmented. The usual locations of the lesions are the wrists, hands (palms), fingertips, knuckles, shoulders, buttocks, feet (heel, toes, soles), knees, ankles and elbows. The buccal mucosa may be involved. In some, after repeated attacks the skin later develops superficial, onion-skin like cicatrices. In

tion occurs in a variety of conditions. Among these are tuberculosis, syphilis, leprosy neoplasms, cicatrices following extirpation of lymph nodes, phlebitis (phlegmasia alba dolens) lymphogranuloma venereum, and ulcer of the leg.

Congenital Elephantiasis (Elephantiasis Telangiectodes) Telangiectatic elephantiasis is a developmental hypertro-

phy of congenital origin. The involved tissues and blood vessels enlarge progressively beginning shortly after birth. The underlying conditions grouped here may belong to lipomatous, fibromatous, or vascular hypertrophies, and may involve the extremities, ears, eyelids, or tongue. Trophedema of Milroy Meigs is discussed elsewhere (see p 761).

EPITHELIOMA ADENOIDES CYSTICUM

SYNONYMS *Acanthoma adenoides cysticum*, *trichoepithelioma papulosum multiplex*, *multiple benign cystic epithelioma*.

Epithelioma adenoides cysticum is characterized by the appearance of flesh-colored, pinkish, translucent round or oval nodules, the surface of which may have a slight central depression.

Incidence It occurs more frequently in women than in men and usually appears at puberty. There is a hereditary tendency to the development of this disease and it often occurs in several members of the same family.

Etiology The cause is unknown. Some of these tumors arise from embryonic cell rests, some come from the epidermis, and some from embryonic hair follicles.

Pathology The histopathology of benign cystic epithelioma reveals strands of proliferating basal cells forming cell nests similar to those seen in prickle-cell carcinoma. Some clinicians believe that epithelioma adenoides cysticum, syringocystadenoma and adenoma sebaceum are variants of the same condition.

Symptoms The lesions as a rule are symmetrically numerous, although solitary lesions occur. They are of light red or yellow color and vary in size from scarcely perceptible papules to the size

of 5 mm. in diameter and are elevated up to 3 mm. The disease begins as a discrete tiny papule which, after attaining the size of a pinhead or pea, remains stationary. The lesions do not coalesce, although they may be closely set, and the larger lesions may have telangiectatic blood vessels associated. The disease shows predilection for the face, particularly about the eyelids, root of nose, cheeks, and chin. The scalp, neck, and chest are occasionally involved. Subjective symptoms are absent.

Diagnosis The location and chronicity of growths, their onset in childhood, and their presence in several members of the same family serve as aids in diagnosis and distinguish it from *adenoma sebaceum* and *milium*, which it resembles.

Complications Ulceration rarely occurs and when present it indicates a change to malignant basal cell carcinoma.

Prognosis If untreated, the lesions persist for an indefinite period. Lesions may ulcerate and terminate in basal cell carcinoma.

Treatment Therapy by roentgen ray and radium is most effective. Desiccation and cauterization may also be used.

with pendulous fibromas, the condition as a whole suggesting neurofibromatosis. In the other it appears to be a slowly progressive congenital malformation characterized by excessive laxness of the skin which usually develops a long time after birth.

Diagnosis The condition must not be confused with the lax skin which develops after loss of much weight in the skin of the abdominal wall in multipara and after removal of tumors.

Treatment This is surgical when indicated.

Cutis Verticis Gyrate (Unna)

SYNONYM: *Bulldog scalp*.

This is a variety of dermatolysis involving the scalp which is loose and has irregular thickened, and variously sized folds and furrows. In some, the condition appears to be congenital, in others, acquired and inflammatory. It has been observed in idiocy, acromegaly, myxedema, and multiglandular deficiency. Accumulated sebum, desquamated cells, and infection in the depths of the folds cause a fetid odor and itching. Surgical ablation in marked cases may be indicated.

ELEPHANTIASIS

"Elephantiasis," or "the elephantiac state" is a term used to designate a chronic progressive regional affection, characterized by a more or less marked enlargement of the skin and subcutaneous tissues due to inflammation and obstruction of the lymphatic vessels.

Symptoms For a description of the elephantiasic syndrome, see p. 339.

Classification Several etiological factors are concerned in the production of this syndrome. Classification is usually based on these factors, as follows:

- 1 Filarial elephantiasis
- 2 Nonfilarial elephantiasis
 - (a) Primary
 - (b) Secondary
3. Congenital elephantiasis

Filarial Elephantiasis (Elephantiasis Arabum) Elephantiasis is endemic in many tropical and subtropical countries. In many sufferers, *Filaria* (*Wuchereria bancrofti*) are found in the blood or lymphatics. Certain conditions (such as lymph scrotum, chylous hydrocele, chylouria, inguinal adenopathy, lymphangitis) are clearly the result of the presence of these parasites and their embryos. It is not so clear, however, that they are

the actual cause of so-called "filarial elephantiasis." Many patients with filariasis do not have this condition. Some think that elephantiasis results from numerous repeated infections over years of habitation in endemic centers. It is, however, believed by many that the filarial lymphatic obstruction merely predisposes to a streptococcal infection. As a result it is now generally thought that tropical (endemic) elephantiasis and sporadic elephantiasis (pseudoelephantiasis or elephantiasis nostras) are essentially the same. The elephantiasic state is, in most instances, limited to one or both lower extremities or to the genitalia. In any case the hypertrophy often enormous, follows repeated regional erysipeloid attacks with fever and general disturbances exactly as occurs in chronic recurrent erysipelas (p. 339). Treatment is largely limited to surgical ablation of the enlarged structures.

Nonfilarial Elephantiasis—PRIMARY ELEPHANTIASIS See *Chronic Recurrent Erysipelas* p. 339.

SECONDARY ELEPHANTIASIS Elephantiasis from a complicating inflammatory and noninflammatory lymphatic obstruc-



Fig. 233 Erysipelas. In butcher working in hog abattoir
(Courtesy of Dr. Joseph V. Klander)

ERYSIPELAS

SYNONYMS: *St. Anthony's fire* *Rothlauf* *erysipel la rose ignis sacer*

Erysipelas is a contagious and infectious disease of the skin and subcutaneous tissue characterized by a single area of redness and swelling which is sharply defined and extends by continuity. The disease is accompanied by constitutional symptoms.

Varieties Erysipelas migrans or ambulans and chronic erysipelas comprise the varieties of erysipelas.

Incidence Erysipelas has a seasonal incidence. It is commonly seen between January and May and is more frequent among women. Recurrences are common among alcoholics. Incidence is greater in infancy and after middle life.

Etiology Erysipelas is due to infection of the skin with the streptococci of Fehleisen or other hemolytic streptococci. It is invariably a wound infection and a history of abrasion is obtainable in the majority of cases. The predisposing causes are unhygienic surroundings, improper food, alcoholism and chronic diseases especially nephritis.

Histopathology The histopathology of erysipelas consists of marked cellular infiltration of the skin. The capillaries are dilated. Chains of streptococci are present in the tissue and beyond the margin of the involved area. The lymph spaces are filled with streptococci.

Symptoms The period of incubation varies from a few hours to seven days. Constitutional symptoms appear suddenly and consist of chills, fever, malaise, headache, anorexia, and vomiting. The temperature ranges between 102 and 106 F (38.6 and 41.1 C) with morning remission and evening exacerbation. Toxic symptoms include the typhoid stage with a dicrotic pulse, diarrhea, delirium and coma. Sudden elevation of

temperature signifies new invasion of the body surface. Constitutional symptoms vanish with the disappearance of the cutaneous lesions.

The cutaneous manifestation starts as a bright red, glazed patch, the size of a



Fig. 232 Erysipelas. Of face. Unusually severe local reaction with bullous lesions here and there over the erysipelatous area. Such bullae are sometimes the result of the application of substances to which the skin is sensitive, such as the mercurials and tincture of iodine.

dime or a silver dollar, sharply defined and elevated above the normal surface of the skin due to the accompanying swelling and edema. The swelling becomes extensive when loose tissue, such as found in the eyelids, is involved. Vesicles and bullae may form on the surface. The reddened lymphatic vessels are occasionally traceable from the site of involvement to the regional lymph nodes which are swollen.

The erysipelatous patch spreads at the periphery and occasionally clears in the center. This spread may be slow or rapid and usually ceases to spread after a few days. The attack runs its course from ten days to three weeks when the edema subsides, the tense, glazed skin pales and slight desquamation ensues.



Fig. 231 Erysipelas. (Courtesy of Dr. A. C. LaBorrette.)

A y part of the cutaneous surface may be involved with erysipelas, although the face is the common site of its involvement. Erysipelas of the face seldom passes down below the chin because of the firm adherence of the subcutaneous tissue in this area.

Erysipelas *ambulans* or *migrans* describes that form of the affection which, after involving a given area, clears and reappears rapidly in neighboring, or even distant, parts.

Subjective symptoms consist of a feeling of local heat, slight itching, and pain. Pain becomes very pronounced if the

scalp is involved. The mucous membranes are rarely affected.

Complications. Subcutaneous abscess may occur over the site of erysipelatous patches especially on the upper eyelids, scalp, forehead, and extremities. The site of erysipelas may occasionally become gangrenous. This condition is usually preceded by the presence of numerous bullae. Death may ensue from septicæmia, septic pneumonia, meningitis, nephritis, or myocarditis.

In infants, erysipelas has a marked tendency to migrate from one part of the body to another. It is often fatal in the newborn because of want of phagocytosis. Phagocytosis precedes in a normal course after the age of three months when the outlook becomes more favorable. A leucocytosis ranging from ten to twenty thousand occurs in mild cases of erysipelas and one ranging from twenty-five to forty thousand in severe cases.

Diagnosis. Dermatitis venenata, acute erythematous eczema, acne rosacea, lupus erythematosus, tuberculosis, and syphilitic lymphadenitis are not to be confused with mild cases of erysipelas. These conditions are not associated with fever and other constitutional symptoms.

The clinical course of erysipelas is so characteristic that there is little risk of error in diagnosis.

Prognosis. Most cases of erysipelas recover. The disease confers little permanent immunity and it has a tendency to recur. Such recurrences are reactions to the streptococci which are present in some obscure focus of infection or may be due to a reinfection at a time of lowered resistance when sufficient antitoxin is absent in the system. Erysipelas is often fatal in infants, the aged, and in alcoholics.

Treatment. *Specific Treatment.* Penicillin in 40,000 to 30,000 units intra

The erysipelatous patch spreads at the periphery and occasionally clears in the center. This spread may be slow or rapid and usually ceases to spread after a few days. The attack runs its course from ten days to three weeks when the edema subsides, the tense, glazed skin pales, and slight desquamation ensues.



Fig. 224 Erysipelas. (Courtesy of Dr. A. C. LeBoeuf.)

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Treatment. *Specific Treatment.* Penicillin in 20,000 to 30,000 units intra-

muscularly every three hours for five days has been used with prompt benefit. *Polyvalent antistreptococcus serum* is the recognized specific for erysipelas, although better results are usually obtained by *hemolytic streptococcus serum* administered intramuscularly in doses ranging from 10 to 20 cc., and repeated if necessary in twelve to twenty four hours. Most successful results attend early administration of these sera. Anaphylaxis is guarded by giving a test hypodermic injection of 0.1 cc. The full dose is then given intramuscularly when no reaction takes place after five minutes. Sera may be given intravenously in severe cases. This treatment may be associated with an initial chill and a rise in temperature, after which the fever subsides. An additional full dose is given intramuscularly the following day to complete the serum treatment. Serum sickness develops any time from a few days to a month in about 20 per cent of cases and can usually be controlled with one of the new antihistamines, benadryl or pyribenzamine.

Good results have been reported from intergluteal injections of milk. The milk is sterilized for ten minutes and the dosage is 5 cc. Aolan (a detoxicated milk protein) has been used with success.

Patients not responding to serum injections are given daily intramuscular injections of 10 minims (1 cc.) of a 1 per cent solution of butyrate of manganese.

Internal Treatment Quinine in doses ranging from 2 grains (0.1 gm.) to 5 grains (0.3 gm.) is given three times each day with 15 minims (1 cc.) of dilute hydrochloric acid. Quinine sulfate in doses of 10 grains (0.6 gm.) may be administered freely during the first few days of severe erysipelas and it has many advocates.

Tincture of iron perchloride administered in three daily doses ranging from 10 to 30 minims (0.6 to 2 cc.) is still regarded by many clinicians as a specific.

The recognized treatment today is the administration of *sulfanilamide* *sulapyridine* or *sulfathiazole*. Adults are given 60 grains of sulfanilamide divided into four doses the first day followed the second day by 40 grains divided into three doses, and continuing the second day's doses until the symptoms have disappeared.

The constitutional symptoms are treated symptomatically.

Local Treatment *Tray therapy* is now regarded valuable in erysipelas. Small patches disappear within twenty four hours, while large patches show limiting of the infection with their gradual disappearance in from two to three days after treatment. The results are obtained by small doses of the unfiltered rays, repeated each day for several days.

Erythema doses of *ultraviolet irradiation* are perhaps just as good as roentgen therapy and one or two irradiations are often sufficient.

Cooling compresses such as a saturated solution of magnesium sulfate or a boric acid solution, are comforting to the patient. Equal parts of ichthyl and water (ichovar) painted on affected areas is useful in controlling the disease. The ichthyl treatment dries into a firm varnish when dusted with a bland powder such as talcum or starch. The unaffected skin just beyond the spreading edge of an erysipelatous patch is often painted with *tincture of iodine* with the hope of increasing leukocytosis in this area. Plain collodion, olive oil, lead water with laudanum, and dusting powders have been satisfactorily employed.

Treatment of Erysipelas in Infants. Large doses of ultraviolet light are

considered by Starr superior to serum therapy in treating infantile erysipelas. Statistical evaluation of the results of treatment by serum and by ultraviolet light indicates that age is an important consideration in determining the type of treatment for erysipelas.

Ultraviolet light produces the best results in infants ranging in age from four to twelve months. Serum can also be used to supplement this treatment, but it is of doubtful value.

Both light and serum therapy and blood transfusions are employed in infants under four months of age. Ultraviolet light reduces the days of morbidity and the mortality rate.

Chronic Recurring Erysipelas

SYNONYMS Chronic recurrent lymphangitis with or without elephantiasis, chronic lymphangitis, solid edema / face and lips, pseudoelephantiasis.

There is a chronic attenuated variety of streptococcal lymphangitis which, after several previous characteristic attacks of erysipelas, does not present the raised red border run the acute course, is accompanied by only slight or no systemic disturbance, and which may eventuate in chronic progressive enlargement of a part.

Etiology The infection generally begins in a recurrent fissure or erosion through which the streptococcus gains entrance. The organisms either lack the usual virulence of the streptococcus of Fehleisen or the individual's resistance to the organism becomes somewhat greater with each attack. Focal infective areas about the mouth, ear, eyelid, and toes may be predisposing factors. The condition is usually seen on the face (lips, eyelids, ears), the lower extremities (foot, leg, or entire limb) and genitalia (penis, scrotum, labia majora).

Symptoms and Pathology The first attack begins in the usual manner in subsequent attacks, in those that will develop pseudoelephantiasis phenomena, the part instead of returning to normal size is larger than after the previous attack. The surface of the skin remains smooth and there is no palp-



Fig. 235 Elephantiasis Nostrae (solid edema) With thickening of lip producing macrocheilia and due to chronic recurrent lymphangitis.

tion the tense elastic sensation of solid edema. The final clinical picture is that of a reddened, greatly swollen lip, eye lid, ear or leg. The skin over the swollen area is tense, difficult to fold, and is adherent to the underlying tissues. It has a puttylike feel on palpation. The edema may be hard and difficult to pit on pressure (pachyderma or solid edema) or it may be soft and pit easily (penis, scrotum). In the fully defined elephantiasis state the skin surface is shiny or it is irregular and verrucous. Its color varies from a dead white to a brownish red. The elevations are lymphangiectases, which are often covered with a mixture of grayish-blackish scale crusta. The dermis and epidermis are commonly thickened and furrowed. In some, vari-

ously sized papillomatous vegetations develop on the surface together with fissures and superficial or deep ulcers which show slight tendency to heal. The condition persists for years.

Treatment The prevention of recurrent attacks depends on the elimination of all sources of infection—fissures and erosions between the toes, corners of the mouth and in the retroauricular and infra auricular areas must be healed and kept so with 3 to 5 per cent *salicylic acid ammoniated mercury ointment*. The following is also a useful prophylactic application

Iodine (crystals)	1.0
Pot. iodide	2.0
Oil cloves	0.1
Oil of pimento	0.1
90% alcohol (70°)	100.0
II	
Acid salicylic	5.0
Tr. mercuriolat	100.0

For the acute attack cold saturated compresses with *boric acid solution* and 70 per cent alcohol are of benefit. X-ray therapy is beneficial. The real problem is to prevent new attacks and inasmuch as the streptococcus is a common cause repeated subcutaneous injections of *streptococcus toxin* or filtrate is worth trying; however it must be administered over a long period of time. Injections of *pennicillin* may be tried. All cases should be given a therapeutic trial of larger doses of *sulfanilamide* (4 gm [60 grains]) in divided doses on first day and then 2 gm (30 grains) daily for ten or more days. The presence of any infectious foci and fissures in particular should be eliminated. Painting open surfaces with a 10 per cent suspension of *sulfathiazole* is helpful. Treatment is often unsatisfactory in advanced cases although marked enlargement in these pseudoelephantiasis

states is occasionally reduced by repeated injections of boiling water. Nonspecific therapy has been of value in some cases. Traube obtained considerable improvement by means of intramuscular injection of *manganese butyrate*. In persistent superficial infection and recurrent lymphangitis, bacteria may remain viable in the soft tissues for over a year. In these cases early strong nonspecific medication (such as *Coley's fluid* or *typhoid vaccine* intravenously) is indicated. Filtered roentgen ray therapy is at times valuable. Surgical intervention in the form of excision of the superfluous tissue (Kondoleon operation) may be used. However even after the removal of the excess tissue the results may be only temporary.

Method of New and Kirch. New and Kirch treated cases of solid edema of the lips and eyelids by injections of boiling water into the involved parts and irradiation externally over the face. The patient was anesthetized with nitrous oxide and the face covered with a thick coating of petrolatum. A metal syringe was used and a few cubic centimeters of the boiling water was injected into many portions of the involved region. This treatment causes considerable local reaction, so that the following day the part is markedly swollen. In a few days the swelling begins to reduce, and it gradually clears. Treatment with radium is given over the involved region from 3000 to 4000 mg. hours with 2 mm. of lead and 1 inch of wood screening being used.

In some cases one treatment accomplished a great deal in preventing recurrence of the swelling but in others four or five treatments at three-month intervals were necessary. When there is no tendency to recurrence of the swelling for a period of six months, the super-

fiuous tissue of the lips and cheeks may be excised, in order to shape the part to its original size. It is essential first, how-

ever to eliminate the recurring swelling with the injections before attempting the operation.

ERYSIPELOID (ROSENBACH)

SYNONYMS *Erythema scarpat*, *erythema migrans*, *acne erysipelat* in man.

Erysipeloid (Rosenbach) is an erysipelas-like eruption usually occurring on the fingers and hands.

Incidence—It is an occupational disease appearing from May to September more often among fishermen, veterinarians, and handlers of diseased animals or their infected product in the abattoir, laboratorv farm, or fish market.

Etiology Erysipeloid is caused by the *Erysipelothrix suis* (*Erysip. thrux rhus seopathiae*) a rod-shaped gram-positive nonmotile organism.

Pathology The capillaries appear dilated and the lymph spaces are filled with lymphocytes. Few polymorphonuclear leukocytes are seen. Bacilli are found in the deep tissues.

Symptoms Erysipeloid is characterized by a smooth circumscribed more or less edematous plaque or a bluish or purplish wheal with elevated irregular border not unlike a lesion of *erythema multiforme*. The plaque extends over the finger and may involve the dorsal surface of the entire hand. A purpuric spot is often seen at the center of the lesion. The sites of predilection are the hands and portions thereof it may appear in the form of paronychia. The face, ears, nares, thighs, and feet have been involved. Occasionally the deeper tissues, including the tendon sheaths and joint capsules, are involved, in which case tenderness and pain are symptoms.

The usual subjective symptoms are itching and burning sensations, although in some cases constitutional symptoms

are present. Lymphangitis and adenitis are occasionally associated.

The incubation period is from one to several days. The eruption usually disappears in a few weeks although recurrences and joint pains may appear as late as five months.

Diagnosis The history of contact with fish putrid materials, or diseased hogs should establish a diagnosis. The process is limited to one location, usually the hands, while *erythema multiforme* is bilateral and the lesions are usually present on other parts of the body. Intracutaneous injection of the causative organism of erysipeloid killed at 70° C. will produce local reaction if the disease is present. Agglutination tests are diagnostic.

Examination of a biopsy specimen is necessary for identification of the organism.

Prognosis The eruption usually lasts several weeks and then disappears spontaneously.

Treatment *Sulfanilamide* and *sulfathiazole* have been successfully used. *Penicillin*, 30,000 units intramuscularly every three hours (total of 1,000,000 units) is curative. The use of an immune serum made from the organism will promptly control the symptoms and cure the infection. The affected part should be immobilized and a 25 per cent *salicylic acid* plaster should be applied. Hot, wet dressings of boric acid are equally helpful. X-ray therapy is of benefit. Surgical incision and drainage may be necessary

ERYTHEMA AB IGNE

SYNONYMS: *Ephelis ab Igne*

Erythema ab igne belongs to a group of erythemas resulting from exposure to heat. It occurs usually on the anterior aspect of the body especially on the legs of stokers, puddlers, glass blowers and blacksmiths.

Pathology The histological picture shows a moderate degree of parakerato-

by a mottled, reticular brown pigmentation. Chronic cases become permanently indurated and assume reddish-brown coloration. The temperature of pale areas of the skin is slightly higher than that of darker areas.

Diagnosis It must not be confused with the following *Livedo reticularis*

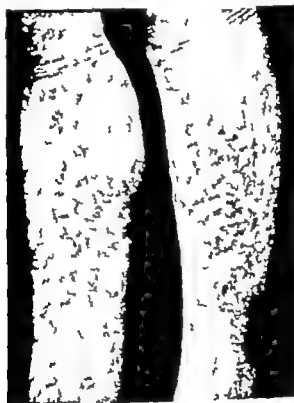


Fig. 236 Erythema Ab Igne

sis in the epidermis and a few polymorphonuclear leukocytes being between the cells of the granular layer. In the corium a moderate amount of cellular exudate is seen in the neighborhood of blood vessels and the coil glands. Brown granular pigment is deposited in the basal cells of the rete mucosum.

Symptoms The condition is characterized by varying degrees of erythema and edema of the skin. This is followed

(*cutis marmorata*) which is seen usually on the legs and arms, especially when the skin is chilled as a bluish mottling. It is due to passive congestion. *Inflammatory livedo reticularis*. In ordinary *cutis marmorata* the changes in the skin are purely in color transient or persistent lived mottling. It occurs especially in children, in those with tuberculosis and chilblain circulation and usually on exposure to cold. In inflammatory *livedo reticularis*, the

reticulations show slightly raised, bluish red, palpably infiltrated lesions of varying extent. The patients are victims of cutis marmorata but develop inflammatory lesions in the markings of this condition. Syphilis and tuberculosis may

select the network of cutis marmorata for their manifestations.

Prophylaxis Prophylaxis consists of a change of vocation.

Treatment Erythema ab igne does not respond to any treatment



FIG. 237 Erythema Ab Igne. I. Remanentary with syphilitic basis. After antisyphilitic therapy the inflammatory phenomena disappeared, leaving only the pigmentary remains characteristic of erythema ab igne.

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Fig. 240 Erythema Multiforme. In female age forty Occurred every spring and was associated with slight fever



Fig. 241 Erythema Multiforme. Left Occurring every spring for six years. Right Balloon type of hands, associated with hyperinflammation.

ERYTHEMA MULTIFORME

SYNONYMS: *Erythema exudativum multiforme*

Erythema multiforme is an acute inflammatory disease of the skin characterized by a varicolored polymorphous rash and exudation into the deeper layers of the integument.

Incidence The disease occurs at any age attacking females more frequently

fish and other foods—a fact suggesting the absorption of products of decomposition. The disease has also been associated with focal infection of the teeth, tonsils, and accessory air sinuses. It is also possible that a number of infections may produce the symptom complex known as erythema multiforme.

Histopathology The histopathology of erythema multiforme consists of vascular distention with leukocytic perivascular cellular infiltration and the occasional presence of red blood cells. The collagen fibers appear swollen, transparent and stain poorly.

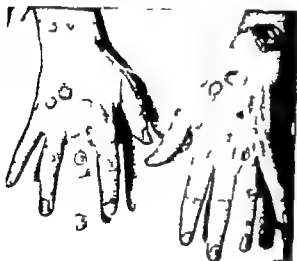


Fig. 238 Erythema Irid. Occurring every year for several years.

than male. It is often met with in spring and autumn. Annual recurrences are common.

Etiology The disease is obscure and constitutes about 1 per cent of dermatologic cases. The symptomatology suggests the presence of toxin in the circulation. It is, therefore, considered a manifestation of streptococcal toxemia. Some clinicians regard the lesions as tuberculids or dermatophytids. Lesions may be produced by hematogenous dissemination of the tubercle bacilli or by the trichophyton fungus in connection with allergic hypersensitivity of the integument. Serum rashes are regarded as a form of erythema multiforme. Erythema multiforme has been reported to occur after the ingestion of stale meat,



Fig. 239 Erythema Multiforme Bullosum. (Courtesy of Dr. Jacques P. Guequierre.)

Forms The term "erythema multiforme perstans" identifies a form of erythema multiforme bearing the physical characteristics of erythema multiforme and urticaria. There is, however, a pre-

The mucous membranes of the mouth, tongue, pharynx, and conjunctivae may be involved.

Diagnosis The diagnosis of erythema multiforme is made from the history of the disease and the multiform character of the lesions.



Fig. 213 Erythema Multiforme
Nodular type which developed after
"ala spora."

The maculopapular type should be distinguished from maculopapular syphiloderm, urticaria and drug eruption. Syphilis is ruled out by the positive serology, general lymphadenitis, and other characteristic stigmata of the disease. Urticaria is of comparatively short duration and has uniformly sized white lesions. Drug eruptions are more generalized in distribution and the history reveals the offending drug employed by the patient.

Annular lesions of erythema multiforme can be confused with pityriasis rosea and ringworm. The history and duration of the disease rule out both conditions.

Vesicular and bullous types of erythema multiforme should be differentiated from dermatitis herpetiformis and pemphigus. *Dermatitis herpetiformis* is a chronic intermittent disease, while erythema multiforme is acute and dermatitis herpetiformis is characterized by intense itching which is often followed by scarring. The bullae of pemphigus arise from the normal integument, a phenomenon which is diagnostic of this disease.

Lupus erythematosus may be confused with erythema multiforme. Erythema multiforme is differentiated from lupus erythematosus by its acute course and the fact that it is never acaly.



Fig. 214 Erythema Multiforme
(phenolphthalein) Eruption on glans
penis. The lip and mouth were also in-
volved.

Prognosis Erythema multiforme is a self-limiting disease which regresses in ten to thirty days.

Erythema multiforme is of grave significance in pregnancy when it may necessitate abortion.

Erythema Multiforme

dominance of one type of lesion over the other. Descriptive terms such as "erythema papulatum," "erythema tuberculatum," "erythema vesiculosum," "erythema annulare," and "erythema marginatum" are used to identify various types of erythema multiforme.

Erythema iris, or herpes iris, is a form of erythema multiforme in which the lesions have a distinctive form. A typical lesion of herpes iris exhibits a central

the orifices (mouth, nose, urethra, vagina, anus, and conjunctiva). Klapaer considers it a form of erythema multiforme.

Symptoms. The rash in erythema multiforme may be preceded by prodromal symptoms consisting of gastroenteritis, coryza, malaise, joint and muscle pains, or tonsillitis. The rash becomes evident twelve to twenty-four hours after these prodromal symptoms. The eruption is, as a rule, of sudden onset. The rash



Fig. 242 Erythema Multiforme. Occurring every spring for six years.

bullae surrounded by an erythematous zone. Occasionally more than one concentric zone surrounds each bulla and in this case each zone is paler than its neighbor. The central bulla or vesicle may not be very distinct, but the concentric rings are plainly discernible. This type of eruption usually occurs on the back of the hands, wrists, and forearms.

"Ectodermosis erosiva pluriorificialis" (Stevens-Johnson disease) has been described as characterized by acute onset, fever, constitutional symptoms, a polymorphic but especially vesicular eruption (notably of the hands and feet) accompanied by inflammation of some or all of

consists of erythematous patches of irregular outline varying in form and size. Patches may consist almost entirely of small and large flattened papules or tubercles. Lesions may be macular, papular, nodular, urticarial, vesicular, bullous, and purpuric, and they may be mixed. Patches may appear more frequently on the dorsum of the hands, feet, forearms, and legs; less frequently on the neck and cheeks; infrequently on the trunk. The accompanying exudation may be so marked that vesicles and bullae are formed (erythema bullosum). The skin of the wrists and other joints is the common site for these bullae and vesicles.

ERYTHEMA NODOSUM

SYNONYM *Dermatitis centraliformis.*

Erythema nodosum is an acute inflammatory and painful disease of the skin characterized by the appearance of rounded or oval nodules, usually on the pretibial surface of the legs and extensor surface of the forearms.

Incidence The disease occurs during the spring and fall (rarely in summer) and affects young adult females more often than males.

Etiology Probably the most common cause of erythema nodosum is a micro-organism which has been identified as a diplococcus. It has occurred in epidemic form and is then called nodal fever.

Tuberculosis is also a causative agent and this is especially important in children. The tuberculin reaction is usually negative before the lesions appear but becomes positive after their appearance.

The following infectious diseases are occasionally associated with erythema nodosum: measles, German measles, scarlet fever, chickenpox, smallpox, diphtheria, whooping cough, mumps, influenza, typhoid and typhus fever, epidemic meningitis, malaria, trypanosomiasis, gonorrhea, chancre, primary and secondary syphilis, and septic sore throat. It is also often associated with rheumatism, chronic tuberculosis, endocarditis, and pericarditis. Foci of infection (teeth and tonsils) have also been incriminated. Drugs, especially iodides, bromides, salicylates, mercury, arsenphenamine, antipyrine, phenacetin, sulfonamides, withyol, ipecac, and diphtheria antitoxin are occasionally the etiological factors. Phlyctenular conjunctivitis is also often associated with this disease.

Pathology: The vessels of the cutis and of the papillae are dilated and sur-

rounded by a narrow band of dense infiltration consisting of lymphocytes and polymorphonuclear leukocytes. The cross-section appearance of these vessels is characteristic of this affection. In the



Fig. 346 Erythema Nodosum.

vessels and particularly in the veins, thrombuslike collections of white blood cells are a common finding. The epithelium and endothelium show signs of proliferation. The collagenous tissue immediately surrounding the infiltrated

Treatment. General Treatment Intestinal toxemia is treated by proper diet and the use of *intestinal antiseptics*. The diet should be nourishing. Red meat and articles of food rich in purine bodies are forbidden. The *salicylates* are administered in large doses. *Colloidal*

Local Treatment Burning and itching are relieved by *calamine lotion* to which 1 per cent *phenol* and 2 per cent *schikol* have been added.

Bullae when present are opened by a sterilized needle and dressed with *Lassar's paste*.



Fig. 215 Erythema Multiforme Bullon type of hands, associated with hyperinfection.

sulfur and *arsenical preparations* are beneficial.

Mercuric cyanide 0.01 (1/100 grain) administered intramuscularly or intravenously every two or three days for about six injections is of definite help; it shortens the duration of this disease.

The *sulfonamides* and *penicillin* have been of value in some febrile forms.

Mouth lesions are satisfactorily treated by *tincture of kino*, *tincture of krameria*, *tincture of myrrh* or by an *alkaline mouthwash*.

Wet dressings of *potassium permanganate* (1:5000) and a *tannic acid dusting powder* are advised for *scrotal lesions*.

Therapy by *ultraviolet light* and *x ray* is of benefit.

Prognosis The duration of erythema nodosum is from two to six weeks. Recurrences are rare.

Prophylaxis Recurrences are prevented by wearing woolen underclothing and flannel bandages around the joints. Avoidance of wind and cold and abstinence from strenuous exercise are prophylactic measures.

Treatment Occlusive alcohol dressings are beneficial and promote regression of the lesions. The application of

dry heat is also helpful. If febrile symptoms are present, rest in bed is advisable. *Strontium salicylate* in daily dosage of 3.0 to 6.0 is an acceptable procedure. If pain in the joints is pronounced, the following treatment is advised.

Morstan.	50 to 60.
Ol. ricini	5.0
Lubol.	100
Petrolatum q. s. ad.	300
Use. Rub on painful joints once each day	

ERYTHEMA PALMARE (LANE)

SYNONYM *Red palms.*

This condition is not uncommon. It is characterized by a symmetrical and marked erythema of the palms, sharply limited at the wristline and sides of the hands.

Etiology It is seen in the male and female and in a variety of both normal

and abnormal conditions. It may be congenital and familial, and has been observed in pregnancy cirrhosis of the liver pulmonary tuberculosis, syphilis, and hyperthyroidism. Capillary studies by Walsh and Becker usually showed a dilatation of the venous side of the cap-



Fig. 242 Erythema Palmare. (Courtesy of Dr. Henry Beckus.)

areas is usually edematous. The epithelium is only secondarily involved. If the infiltrating nodes extend upward the cells may become atrophic. In the latter stages, a diffuse infiltrate is found involving the cutis forming small nodes in the fatty tissue beneath the cutis.



Fig. 247: Erythema Nodosum.

The picture is not uniform or diagnostic; however, one may be certain that the affection is due to something carried via the blood, as all the pathological changes are limited to the vessels themselves and to their immediate neighborhood.

Symptoms. Erythema nodosum is usually symmetrical and appears acutely on extensor surfaces of the legs. Lesions occasionally appear on the forearms, thighs, gluteal regions, the trunk, and rarely over the face. The affection is characterized by the appearance of red,

tender, sometimes painful nodules blending into the neighboring skin with surrounding edema. They enlarge to about 2 cm. in diameter and remain stationary. They do not coalesce to form plaques. There may be few or as many as thirty lesions. In the course of a few days they become flatter, livid, and undergo color changes similar to those seen in hematomas such as brown, yellow and green, hence the term "dermatous contusions." In a few weeks they disappear without leaving scars; however, slight hyperpigmentation and slight scaling may result. New lesions may appear and the affection may continue for five or six weeks longer.

Prodromal symptoms, such as slight hyperpyrexia, chills, malaise, and anorexia are often present. Sore throat, muscle and joint pains often accompany the affection. Gastrointestinal symptoms are likely to occur in children.

Diagnosis. Symmetrical, tender erythematous nodules on the extensor surfaces of the legs are pathognomonic of this affection. *Pyogenic nodules* are usually unilateral and suppurative. *Erythema induratum* is localized on the posterior surface of the lower third of the leg when the lesions are more persistent, eventually ulcerating. *Syphilitic gummata* are unilateral, painless, and more sharply defined and more prone to ulceration. *Erysipelas* is more acute, diffuse, and superficial than erythema nodosum. The lesions following the use of iodides and bromides disappear on withdrawal of the drug.

Complications. Suppuration of the nodes occasionally though rarely occurs. Mucous membranes are seldom affected and visceral involvement is also rare. Pericardial and endocardial involvement has been reported, however this is also rare.

Prognosis The duration of erythema nodosum is from two to six weeks. Recurrences are rare.

Prophylaxis Recurrences are prevented by wearing woolen underclothing and flannel bandages around the joints. Avoidance of wind and cold and abstinence from strenuous exercise are prophylactic measures.

Treatment Occlusive alcohol dressings are beneficial and promote regression of the lesions. The application of

dry heat is also helpful. If febrile symptoms are present, *rest* in bed is advisable. *Strontium subcaps* in daily dosage of 3.0 to 6.0 is an acceptable procedure. If pain in the joints is pronounced, the following ointment is advised

Messian	20 to 40.
Oil ricin	100
Lanolin	100
Petrolatum q. s. ad.	200
Scr. Rub on painful joints once each day	

ERYTHEMA PALMARE (LANE)

SYNONYM *Red palms.*

This condition is not uncommon. It is characterized by a symmetrical and marked erythema of the palms, sharply limited at the wristline and sides of the hands.

Etiology It is seen in the male and female and in a variety of both normal

and abnormal conditions. It may be congenital and familial, and has been observed in pregnancy, cirrhosis of the liver, pulmonary tuberculosis, syphilis, and hyperthyroidism. Capillary studies by Walsh and Becker usually showed a dilatation of the venous side of the cap-



Fig. 248 Erythema Palmare (Courtesy of Dr. Henry Beckus.)

areas is usually edematous. The epithelium is only secondarily involved. If the infiltrating nodes extend upward the cells may become atrophic. In the latter stages a diffuse infiltrate is found involving the cutis forming small nodes in the fatty tissue beneath the cutis.



Fig. 217: Erythema Nodosum.

The picture is not uniform or diagnostic; however one may be certain that the affection is due to something carried via the blood, as all the pathological changes are limited to the vessels themselves and to their immediate neighborhood.

Symptoms. Erythema nodosum is usually symmetrical and appears acutely on extensor surfaces of the legs. Lesions occasionally appear on the forearms, thighs, gluteal regions, the trunk, and rarely over the face. The affection is characterized by the appearance of red

tender sometimes painful nodules blending into the neighboring skin with surrounding edema. They enlarge to about 2 cm. in diameter and remain stationary. They do not coalesce to form plaques. There may be few or as many as thirty lesions. In the course of a few days they become flatter, livid, and undergo color changes similar to those seen in hematomas, such as brown, yellow and green, hence the term "*dermatosis contusiformis*." In a few weeks they disappear without leaving scars; however slight hyperpigmentation and slight scaling may result. New lesions may appear and the affection may continue for five or six weeks longer.

Prodromal symptoms, such as slight hyperpyrexia, chills, malaise, and anorexia are often present. Sore throat, muscle and joint pains often accompany the affection. Gastrointestinal symptoms are likely to occur in children.

Diagnosis. Symmetrical, tender erythematous nodules on the extensor surfaces of the legs are pathognomonic of this affection. *Pyogenic nodules* are usually unilateral and suppurative. *Erythema induratum* is localized on the posterior surface of the lower third of the legs when the lesions are more persistent, eventually ulcerating. *Syphilitic gummata* are unilateral, painless, and more sharply defined and more prone to ulceration. *Erysipelas* is more acute, diffuse, and superficial than erythema nodosum. The lesions following the use of *iodides* and *bromides* disappear on withdrawal of the drug.

Complications. Suppuration of the nodes occasionally though rarely occurs. Mucous membranes are seldom affected and visceral involvement is also rare. Pericardial and endocardial involvement has been reported, however this is also rare.

Prognosis The duration of erythema nodosum is from two to six weeks. Recurrences are rare.

Prophylaxis Recurrences are prevented by wearing woolen underclothing and flannel bandages around the joints. Avoidance of wind and cold and abstinence from strenuous exercise are prophylactic measures.

Treatment Occlusive alcohol dressings are beneficial and promote regression of the lesions. The application of

dry heat is also helpful. If febrile symptoms are present, rest in bed is advisable. *Strontium salicylate* in daily dosage of 3.0 to 6.0 is an acceptable procedure. If pain in the joints is pronounced, the following ointment is advised

Menthol	20 to 40
Oil rose	50
Lanolin	100
Petrolatum q. s. ad	200
See Rub on painful joints once each day	

ERYTHEMA PALMARE (LANE)

SYNONYM *Red palms.*

This condition is not uncommon. It is characterized by a symmetrical and marked erythema of the palms, sharply limited at the wristline and sides of the hands.

Etiology It is seen in the male and female and in a variety of both normal

and abnormal conditions. It may be congenital and familial, and has been observed in pregnancy, cirrhosis of the liver, pulmonary tuberculosis, syphilis, and hyperthyroidism. Capillary studies by Walsh and Becker usually showed a dilatation of the venous side of the cap-



Fig. 248 Erythema Palmare. (Courtesy of Dr. Henry Beckus.)

areas is usually edematous. The epithelium is only secondarily involved. If the infiltrating nodes extend upward the cells may become atrophic. In the latter stages, a diffuse infiltrate is found involving the cutis forming small nodes in the fatty tissue beneath the cutis.



Fig. 217 Erythema Nodosum.

The picture is not uniform or diagnostic; however, one may be certain that the affection is due to something carried via the blood, as all the pathological changes are limited to the vessels themselves and to their immediate neighborhood.

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tender sometimes painful nodules blending into the neighboring skin with surrounding edema. They enlarge to about 2 cm. in diameter and remain stationary. They do not coalesce to form plaques. There may be few or as many as thirty lesions. In the course of a few days they become flatter, livid, and undergo color changes similar to those seen in hematomata, such as brown, yellow and green, hence the term "*dermatosis contusiformis*." In a few weeks they disappear without leaving scars; however, slight hyperpigmentation and slight scaling may result. New lesions may appear and the affection may continue for five or six weeks longer.

Prodromal symptoms, such as slight hyperpyrexia, chills, malaise and anorexia are often present. Sore throat, muscle and joint pains often accompany the affection. Gastrointestinal symptoms are likely to occur in children.

Diagnosis. Symmetrical, tender erythematous nodules on the extensor surfaces of the legs are pathognomonic of this affection. *Pyogenic nodules* are usually unilateral and suppurative. *Erythema induratum* is localized on the posterior surface of the lower third of the leg when the lesions are more persistent, eventually ulcerating. *Syphilitic granulata* are unilateral, painless, and more sharply defined and more prone to ulceration. *Erysipelas* is more acute, diffuse, and superficial than erythema nodosum. The lesions following the use of iodides and bromides disappear on withdrawal of the drug.

Complications. Suppuration of the nodes occasionally though rarely occurs. Mucous membranes are seldom affected and visceral involvement is also rare. Pericardial and endocardial involvement has been reported, however, this is also rare.

Among them are *pellagra* in which a persistent erythema involving the uncovered parts is often the first sign of the vitamin deficiency (see p 381) *neurosyphilis* (Unas) a persistent, almost fixed, annular or polycyclic pink, slightly scaly nonpruritic and noninfiltrated erythema observed in chronic syphilis and extremely resistant to therapy (Fig. 508) *erysipelas perstans* (Kaposi) a form of acute disseminated lupus erythematosus (see p 790) *leptoides* (see p 441) *purpura* (see p 550) *seborrheic dermatitis* (see p 224) *erythema elevatum diutinum* (see p 788) initial erythema of mycosis fungoides (see p 814) toxic erythemas (due to foods, drugs, infections, and their toxin) Polycyclic urticarial erythemas and *dermatitis herpetiformis* also must be excluded from this rare group.

The following, in addition to those noted above, appear to be separate clinical states, although several may be varieties of erythema multiforme: In *erythema a vulva centrifuga* (Darier) the lesions, usually on the trunk, are pink to very in color and not scaly and perma-

nent or recurrent, although the actual lesions may run an acute or subacute course. The primary lesion is an urticarial-like spot which quickly becomes annular with cord-like borders. Its chronicity excludes erythema multiforme and erysipelas, absence of pruritus excludes dermatitis herpetiformis, specific studies exclude syphilis and leprosy and histologic studies exclude early mycosis fungoides, *granuloma annulare*, and lichen planus. In *erythema figurata perstans* (Wende) the lesions, usually on trunk or extremities, begin as pink maculopapules which clear in center to form variably sized, slowly or rapidly spreading, circinate or gyrate erythematous patches. The epidermis of the outer lesion border is smooth and slightly raised, on the inner border it is scaly and desquamating. There are no subjective symptoms. *Erythema chronicum migrans* (Lipkecht) and *erythema simplex gyrtatum* (Jadassohn) may be variants of Wende's erythema perstans.

Treatment Usually of no avail but erythema doses of ultra violet radiation have caused some lesions to disappear.

ERYTHRASMA

Erythrasma is a vegetable parasitic disease characterized by slowly evolving brownish, scaly patches in the groin and axillae, and occasionally in other intertriginous regions.

Incidence: It is more common in men than in women and also occurs more often in the obese. It does not occur in children. It is rarely seen in cold temperate climates.

Etiology It is caused by the *Microsporon minutissimum* (*Actinomyces minutissimus*). Some authors believe it to be *Streptothrix* rather than belonging to the genus *Microsporon* (Sabou-

raud). The fungus consists of very fine long, mycelial threads and very minute spores.

Symptoms The clinical findings consist of irregular sharply circumscribed, reddish brown or reddish yellow scaly dry patches. It begins as a dry reddish macule which spreads peripherally. The scales are fine, dry and powderlike. The sites of predilection are the genito-crural axillary regions, and intertriginous areas. Slight itching may be present.

Diagnosis It is differentiated from *tinea versicolor* by the brownish instead of the yellowish color and by the loca-

illary loop and the superficial venous plexus

Symptoms The erythema is sharply defined bright or dark red and largely limited to the skin over the thenar and hypothenar eminences and fingertips. It is aggravated by heat, warm weather and immersion of the hands in hot water. The condition develops gradually and at any age. It varies in intensity from time to time and from patient to patient. Under pressure it temporarily disap-

pears either rapidly or sluggishly. Occasionally the soles are also involved. Telangiectases in varying degree are frequently associated. There are no subjective symptoms.

Prognosis The condition is apt to be persistent, but when it develops during pregnancy the erythema tends to fade after parturition.

Treatment Therapy is usually not necessary. If requested a simple astringent lotion may be prescribed.

ERYTHEMA PERSTANS

Under a number of terms, rare instances of persistent or recurrent circumscribed types of inflammatory erythema of undetermined etiology with or without scaling or infiltration have been described. There are however a number of

well-defined dermatoses in which scaly or nonscaly pruritic or nonpruritic, slightly infiltrated or noninfiltrated, fixed or slowly extending persistent erythemas occur. Clinically and generally etiologically most of these have been evaluated.



Fig. 249 Erythema Chronicum Migrans (Lipschitz)

The shape of the individual lesions varies, some being circinate, and others oval or oblong, with a jagged or irregular outline. Their size varies. The walls of the vesicles are thin, easily ruptured, and translucent. Many of them are surrounded by a reddish areola. With few exceptions, these are not umbilicated and are usually unilocular. A characteristic feature of varicella is the occurrence of successive crops, four or five of which may appear in the course of an attack, so that at a given moment lesions may be seen in all stages of development.

The contents of the vesicles are at first clear but within twenty-four hours they become opaque, or the vesicles may dry up rapidly before the contents become turbid. Some vesicles may show umbilication as they dry from the center. Finally a scab is formed which, on separation, leaves a pigmented macule. The pigmentation gradually fades, but pale spots may persist for years. The number of lesions varies in different cases from half a dozen or less to several thousand. In mild cases five to ten lesions may be the extent of the eruption. Although varicella does not affect the mucous membranes to the same extent as varola, lesions on the buccal, palatal, and faucial mucosae are not uncommon, where they are usually seen as small gray erosions.

The degree of constitutional disturbance present generally bears a direct relation to the extent of the eruption.

A rare form of chickenpox is that known as "bullous" or "pemphigoid varicella" in which the vesicles are of considerable size from the first or are of normal dimensions at first and subsequently grow to an abnormally large size.

Complications. In the majority of cases, varicella runs an uncomplicated course. The complications that do occur are usually attributed to secondary in-

fection. In the scalp, the lesions are often scratched because of the itching; secondary infection, with enlarged and tender occipital and posterior cervical glands, results. Other secondary infections, such as boils, subcutaneous abscesses, impetigo, and erysipelas, are occasionally ob-



Fig. 251. Varicella. Lesion on palate. (Courtesy of Dr. A. C. Laloe-cetta.)

served. Ocular lesions are observed in cases in which the eruption is most abundant. Laryngeal lesions may necessitate tracheotomy.

Differential Diagnosis. Ordinarily the diagnosis is not difficult. The disease which should be distinguished from a severe case of chickenpox is a mild attack of smallpox. In the latter the eruption is preceded by constitutional symptoms of varying intensity lasting about four days, with an elevated temperature. This recedes upon the appearance of the eruption and rises again upon the development of the vesicles. In varicella, the pro-

tion It is differentiated from *pityriasis rosea* and *tinea cruris* by the absence of inflammation and a different symptomatology

Prognosis The prognosis is favorable however recurrences are common.

Treatment Treatment is identical with that prescribed for *tinea versicolor* .

THE EXANTHEMATA

The eruptive fevers or the exanthemata are viewed by some as conditions borderline between internal medicine and dermatology. Common usage places them in the domain of general medicine. However their cutaneous features are so prominent a part of these diseases that a brief presentation of them and their differentiation from other eruptive diseases is advisable. It might be added that the exanthemata have the following in common (1) They are specific infections and highly contagious (2) they occur most commonly in children (3) they are self limited diseases and (4) one attack usually confers permanent immunity

Chickenpox

SYNONYM *Varicella* .

Definition This is an acute contagious disease characterized by an erythematovesicular eruption which appears in successive crops.

Incubation Period It is usually four to sixteen days

Symptoms In adults, in whom the pre-eruptive stage is often longer than in children, mild fever a feeling of malaise, anorexia headache, backache and vomiting may precede the rash by one or more days. In children the eruption is usually the first sign of the disease.

The eruption first appears on the front and back of the trunk and inner sides of the thigh in the form of papules and vesicles. The papular stage is sometimes very short and, when the patient is first seen vesicles are already fully developed. The face, scalp and proximal ends of the

limbs become invaded within the first twenty four hours and fresh lesions continue to appear for the next two or three days. The total duration of the eruption rarely exceeds eight to ten days. The eruption tends to be more profuse and even confluent in an area of the skin sub-



Fig. 250: *Varicella* . Lesions in various stages of development. (Courtesy of Dr. A. C. LaBoretta.)

ject to irritation e.g. by a pre-existing eruption such as eczema, the application of iodine or a mustard plaster, scratching, or sunburn. In infants, the eruption is particularly liable to be well marked on the buttocks and thighs, owing to the irritation caused by the body excretions, such as urine and feces. The lesions are always thickest on the trunk and proximal ends of the limbs, while on the distal extremities, except in severe attacks and apart from local irritation, they are sparse and frequently absent altogether

noted on the entire cutaneous surface. It is not unusual for the face, chest, and arms to show the eruption at its height, while the legs are unaffected. The lesions consist of pale pink macules, round or irregular in shape, and varying in size from that of a pinhead to a small bean. In some cases, they become distinctly raised, and form irregular blotches, which are impossible to distinguish from true measles. In a less frequent variety the lesions, which at first are discrete, become confluent so that the eruption closely resembles that found in scarlet fever. A previously irritated region or one where pressure has existed seems to increase the intensity of the eruption and to encourage confluence. The circumoral region is usually covered with the lesions. The eruption lasts for an average of two to three days and rapidly fades without leaving the brownish staining characteristic of measles. In some outbreaks, the eruption may be scarcely evident and last only twenty-four hours. In others, the eruption may be protracted in some regions of the body and persist for four or five days.

Desquamation is absent. Catarrhal disturbances are slight or entirely absent.

The fever variations are dependent upon the severity of the attack and the character of the prevailing epidemic. There is usually an insignificant rise in temperature, if there is any fever at all. Adults with German measles present more of a temperature rise than children.

Enlargement of the lymphatic glands has long been regarded as a symptom of considerable diagnostic importance; however, glandular enlargement is also present in scarlet fever and measles.

Differential Diagnosis. A mild case of measles may be confused with an ordinary case of rubella. In measles, there is an incubation period of ten to eleven

days, more marked catarrhal symptoms, the presence of Koplik's spots, and a higher temperature rise for four days before appearance of the eruption, which is darker, crescentic, and coalescent.

Scarlet Fever. A mild case of this disease is often very confusing and difficult to distinguish from rubella. A short incubation period of two to seven days, with initial symptoms of nausea, vomiting, sore throat, headache, and fever followed by a rash which avoids the face, but involves the soft palate, are symptoms indicative of scarlet fever. In rubella the rash covers the face, especially the circumoral regions; in scarlet fever the face is pale.

Drug Rashes. Differentiation will depend upon the disease for which the causative drug was used.

Prognosis. This is more favorable in rubella than in any other acute infectious disease. Complications are uncommon and death is rare.

Treatment. No special treatment is required. Patients should be kept in bed if there is a febrile reaction. Diet need not be restricted unless fever and constitutional disturbance are present.

Measles

SYNDROMES Morbilli, rubella.

Measles is a highly transmissible disease, marked by catarrhal symptoms, fever, and a characteristic eruption, which appears on the fourth day of the disease. It is the most common of all communicable diseases.

Incubation Period. It is usually ten to eleven days.

Prodromal Stage. It lasts four days. The characteristic features of this stage are the gradual rise in temperature, catarrh of the mucous membranes, prodromal rashes, and Koplik's spots. Other symptoms include loss of appetite, head-

dromal symptoms are mild if present, but more often the eruption is the first evidence of the disease. If a rise in temperature is manifested it rarely lasts longer than forty-eight hours and without a secondary rise. In variola the lesions are thicker deeper shot like on palpation not easily broken uniform in distribution and umbilicated. They reach a pustular stage then persist for about fourteen days, finally drying to yellowish crusts. In varicella the vesicles are superficial thin irregular walled easily broken by finger pressure not umbilicated appear in successive crops on an erythematous base and last about eight days. The finding of a recent vaccinal cicatrix would favor varicella.

Impetigo sometimes appears as a secondary infection superimposed upon varicella. In this instance impetigo engrafts itself upon the border of the lesions in the form of vesicular rings. These rapidly change to pustules, burst and form superficial crusts. If impetigo appears as an independent infection it can be distinguished from varicella by the fact that in impetigo the patient suffers from no constitutional symptoms, the lesions change quickly to large superficial, irregular crusts and do not appear in the mouth and throat. The lesions in varicella appear in successive crops and disappear in a short time without specific local therapy.

Prognosis. The disease is mild and ends in recovery in ten to fourteen days. Complications occur rarely and only when such structures as the larynx or eye are involved does the prognosis for complete recovery become guarded.

Prophylaxis. Only in exposed individuals, where a recent pre-existing infection has occurred or in debilitated persons, is prophylaxis indicated. This consists in injecting 10 to 20 cc. of con-

valescent tyophile serum. It will prevent the disease from developing if given before the fifth day of the incubation period and, if given later will modify the disease.

Treatment. Isolation should be maintained until there is complete separation of all scabs. Application of an antipruritic solution locally to each lesion will hasten drying of the lesions, lessen pruritus, and prevent scratching secondary infection, and subsequent scarring. A lotion containing 1 per cent picric acid and 1 per cent iodine in alcohol is excellent. In the final stages daily warm baths with a heavy lather of soap will hasten separation of the crusts.

German Measles

SYNOVIAL Rubella.

Definition. An acute contagious disease characterized by a finely mottled eruption and frequent enlargement of the lymphatic glands with little or no constitutional disturbance.

Incubation Period. It is fourteen to sixteen days and occasionally as long as twenty-one days.

The Prodromal Stage. It is longer in adults than in children and may be accompanied by headache, backache, muscular fatigue, anorexia and slight rise of temperature as well as a slight degree of catarrh, shown by cough, sneezing, nasal discharge, or conjunctivitis. The duration of the prodromal stage varies from a few hours to two or three days.

Eruptive Period. The eruption which is often the first sign of the disease especially in children appears first on the face and then rapidly invades the chest, abdomen, trunk, and finally the extremities. It is not uncommon for the eruption to make its appearance in the reverse to that described above. The maximum intensity of the rash is not simultaneously

week or two and serves to establish a retrospective diagnosis of measles.

With the fading of the eruption, there is a general subsidence of the catarrhal symptoms. The temperature returns to normal and remains so unless a complication makes its appearance. Desquamation is only branny in character and does not form a striking feature of this disease.

A rare eruptive type is seen in hyperpigmented measles. In this, the eruption is more intensely red than in ordinary measles, does not fade on pressure, and retains its brilliant color up to the seventh or eighth day before it begins to fade. This type must not be confused with hemorrhagic measles, a fatal type, characterized by extensive hemorrhages in the skin, subcutaneous tissues, and from the mucous membranes. The hemorrhage may appear in the form of epistaxis, melena, hematemesis, and hematuria. The eruption is ill-developed and soon fades.

Complications. Stomatitis may occur in a few instances. Debilitated children, who have been living under unhygienic conditions, are more likely to suffer from some grade of stomatitis, noma in particular.

Herpes labialis occasionally appears early in the disease.

Impetigo and furunculosis are frequent and troublesome during convalescence, and may keep recurring until the patient's resistance has been reestablished.

Otitis media with or without a purulent discharge, is a severe complication and, in many instances, may spread to the mastoid process. Partial or complete deafness may follow.

Blepharitis and styas are not uncommon and may be followed by corneal ulcerations.

Differential Diagnosis. During epidemics, the diagnosis is made easily. The

infectiousness of measles is highest in the prodromal and eruptive stages, when it is spread by coughing or sneezing. If there is a history of exposure to measles, the disease can be suspected in contacts developing catarrhal symptoms. When the catarrhal symptoms are pronounced and Koplik's spots are evident, there is no question about the eruption that will follow. In the absence of severe catarrhal manifestations and the nonappearance of Koplik's spots, the eruption must be distinguished from other infectious and noninfectious eruptive diseases.

Rubella. In rubella, there is absence of or a very short, prodromal stage. The eruption is the first evidence of the disease, and there are very mild constitutional symptoms and a low temperature of short duration. The eruption consists of pale pink macules which spread rapidly and last only a short time (one to three days) fading without leaving pigmentation. In measles, the symptoms may be mild but a prodromal stage of three to four days is always present. Temperature rise is conspicuous, in a sudden elevation or gradually rising until the appearance of the eruption, when it recedes gradually. The morbilliform eruption slowly fades, leaving a brownish pigmentation which lasts for ten to fourteen days.

Scarlet Fever. The prodromal stage may be marked by gastrointestinal symptoms and punctate erythema of the skin and palate which avoids the face. There is circumoral pallor with a tongue resembling a strawberry in typical cases or a tongue resembling a strawberry coated with cream if nontypical. The eruption fades without leaving pigmentation and in ten to fourteen days desquamation commences. In measles, there are catarrhal symptoms in the prodromal stage, followed by an eruption which ap-

ache and marked drowsiness. The catarrhal signs consist of lachrimation, conjunctival injection, edema of the palpebral conjunctiva (especially of the lower lids), photophobia, nasal discharge (occasionally accompanied by epistaxis), attacks of sneezing and a frequent hoarse cough. Upon inspection the tonsils, pharynx, and palate show a varying degree of congestion. The oral mucous membrane, particularly the gums, is red, dened and swollen.

Koplik's spots are slightly raised, irregular bluish white spots, each about the size of a pinhead, surrounded by a narrow areola of reddened mucous membrane. They are usually situated on the buccal mucosa. Their number varies from a few, three or four on each side of the mouth, to hundreds. They precede the specific eruption by two or three days and disappear usually upon the appearance of the eruption or in its early stage. Koplik's spots are absolutely pathognomonic of measles, being present in 80 to 90 per cent of all cases. The temperature, hitherto normal, ranges from 99 to 104° F (37.2 to 40° C) at night and falls to normal or shows a considerable remission in the morning. Less frequently it remains high throughout the prodromal period, showing no tendency to drop before the appearance of the eruption.

Eruptive Stage. The specific eruption of measles usually appears on the fourth day. The lesions consist of discrete raised dusky red macules which vary in size from a pinhead to a bean. They appear first behind the ears, on the chin and upper lip, and in the next twenty-four hours spread over the face and neck, covering the face more copiously than the rest of the body, which becomes involved soon after. The eruption is thickest and brightest on face

which assumes a bloated appearance. The individual lesions, which are at first small, rapidly increase in size and become distinctly papular and frequently crescentic in shape. In many cases, the lesions tend to become confluent, so that the skin, especially on the back and buttocks, shows



Fig. 252: Rubella. Fading roseola. (Courtesy of Dr. A. C. LaBoccetta.)

a diffuse erythema closely simulating that which is observed in scarlet fever.

During the development of the eruption, there is an aggravation of the local and constitutional symptoms, evidenced by restlessness, insomnia, marked general discomfort, and an increase of the catarrhal signs. The cough becomes more severe and often paroxysmal; the voice becomes hoarse. The eruption usually lasts four or five days, and as a rule, disappears in the same order in which it developed. As it fades, it leaves a brownish pigmentation which may last for a

week or two and serves to establish a retrospective diagnosis of measles.

With the fading of the eruption, there is a general subsidence of the catarrhal symptoms. The temperature returns to normal and remains so unless a complication makes its appearance. Desquamation is only branny in character and does not form a striking feature of this disease.

A rare eruptive type is seen in hyperpigmented measles. In this, the eruption is more intensely red than in ordinary measles, does not fade on pressure, and retains its brilliant color up to the seventh or eighth day before it begins to fade. This type must not be confused with hemorrhagic measles, a fatal type, characterized by extensive hemorrhages in the skin, subcutaneous tissues, and from the mucous membranes. The hemorrhage may appear in the form of epistaxis, melena, hematemesis, and hematuria. The eruption is ill-developed and soon fades.

Complications. Stomatitis may occur in a few instances. Debilitated children, who have been living under unhygienic conditions, are more likely to suffer from some grade of stomatitis, noma in particular.

Herpes labialis occasionally appears early in the disease.

Impetigo and furunculosis are frequent and troublesome during convalescence, and may keep recurring until the patient's resistance has been reestablished.

Otitis media, with or without a purulent discharge, is a severe complication and in many instances, may spread to the mastoid process. Partial or complete deafness may follow.

Blepharitis and sty's are not uncommon and may be followed by corneal ulceration.

Differential Diagnosis. During epidemics, the diagnosis is made easily. The

infectiousness of measles is highest in the prodromal and eruptive stages, when it is spread by coughing or sneezing. If there is a history of exposure to measles, the disease can be suspected in contacts developing catarrhal symptoms. When the catarrhal symptoms are pronounced and Koplik's spots are evident, there is no question about the eruption that will follow. In the absence of severe catarrhal manifestations and the nonappearance of Koplik's spots, the eruption must be distinguished from other infectious and noninfectious eruptive diseases.

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pears first on the face and involves chiefly the circumoral region. The eruption fades gradually after a stay of four to five days and is followed by pigmentation which also gradually disappears.

Smallpox In the prodromal stage, which is of the same duration in both diseases and with similar symptoms in an adult one disease may be easily mistaken for the other. In variola a prodromal morbilliform eruption of smallpox may be mistaken for measles and during an epidemic of smallpox the presence of a morbilliform rash may be wrongly diagnosed as smallpox when actually the case is one of measles. More prominent catarrhal symptoms and Koplik's spots should exclude smallpox.

Typhus Fever and Rocky Mountain Spotted Fever These resemble measles when there is a profuse eruption. The pronounced catarrhal symptoms, with photophobia, lacrimation and Koplik's spots in a prodromal stage of four days, indicate measles.

Common Cold Until more definite evidence occurs, such as Koplik's spots and the characteristic eruption the common cold in non-epidemic times, may be easily mistaken for the catarrhal symptoms initiating measles.

Drug Rash Phenobarbital, dilantin, sulfonamides, bromides, and many other drugs may cause a morbilliform eruption. The absence of a prodromal stage with all the symptoms that usually occur in this period will readily exclude measles.

Prognosis The mortality is highest in the first two years of life. Measles is usually a much milder disease in private practice and in better homes, than in hospitals or in the slums. Anemic, rickety or tuberculous persons are liable to pulmonary or intestinal complications. Bronchopneumonia and otitis media are the commonest complications.

Prophylaxis Prevention or modification of an attack can be obtained by the injection of immune bodies. The extent of modification will depend upon what day of the incubation period the injection is given. The later in the period the immune bodies are injected the less modification of the disease will be obtained. With modification of the disease an active or lasting immunity is established. Complete protection from one exposure to the disease or passive immunity can be afforded if the injection is given before the fifth day after exposure. The preparations used are fresh convalescent serum, adult immune serum, human whole blood, immune globulin derived from human placenta or gamma globulin from human plasma. The usual dose is 10 cc of the serum, 20 to 25 cc of whole blood, or 2 to 4 cc of globulin injected subcutaneously.

Treatment The patient should be kept in bed. The eyes should be bathed frequently with warm boric acid solutions and the lids greased night and morning with boric acid ointment. Because of the photophobia bright light should be excluded or dark glasses or eyeshade worn by the patient. If seen in the pre-eruptive stage an intramuscular injection of 40 cc of convalescent serum or 10 to 20 cc. of gamma globulin will often ameliorate the severity of the disease. In the presence of complications, such as bronchopneumonia or otitis media, the use of sulfadiazine in proper dosage is indicated.

Scarlet Fever

Definition Scarlet fever is an acute infectious disease characterized by an erythematous eruption, fever, lateral angina and a varying degree of constitutional disturbance.

Incubation Period This is usually two to three days, but it may be as long as a week or as short as a few hours.

Symptoms The eruption, which may be the first evidence of the disease, is made up of two elements, an erythema and minute papules or puncta. Usually these two elements appear together or they may appear separately in different

a minute, and then turns pale again remaining pale for two or three minutes.

The eruption is more pronounced in areas that are subjected to warmth or to irritation, e.g. the bends of the elbows, the popliteal spaces, inner sides of the thighs, and in the diaper region of untrained children. It first appears on the neck and upper chest and then spreads to the abdomen and extremities. The exanthem avoids the face; instead the cheeks and forehead are flushed, and the region about the mouth is pale, especially in contrast to the flush on the cheeks. This circumoral pallor is sometimes called Filatow's sign. The duration of the eruption varies from a few hours to four to five days in severe cases. Itching is not infrequently present. The erup-

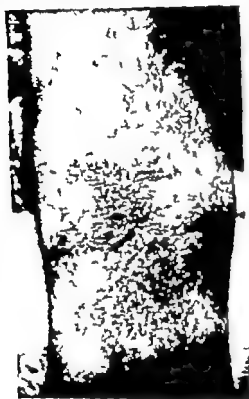


Fig. 233 Scarlet Fever. Second day
(Courtesy of Dr. A. C. LaBorretta.)

part of the body. A diffuse erythema may cover the body and the typical rash occupy only a small area in the bends of the elbows. On the extremities, a morbilliform eruption may be present but a careful inspection of the body especially of the pubes, will reveal an area of punctate erythema. Stroking the skin covered by the eruption will elicit a secondary pallor; a central red line with pale lines on either side or a pale line which is in a minute, becomes red lasting

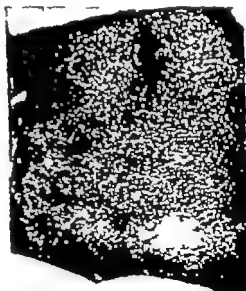


Fig. 234 Scarlet Fever. Desquamation.
(Courtesy of Dr. A. C. LaBorretta.)

tion disappears in the same order in which it made its appearance; therefore if it has faded from the chest, it may still be present on the abdomen and lower extremities. It leaves no pigmentation, but the puncta element persists giving the skin

a goose-flesh like appearance. The skin becomes very dry and harsh to the touch. The only other part of the body that is affected is the soft palate and uvula. The amount and intensity of the eruption on the soft palate closely follows the severity of the eruption on the skin.

During the attack the lips are congested and bright red. This redness disappears at the end of a week when the lips become grayish with a tendency to fissuring especially at the commissures.

The tongue at onset of the disease is covered with a thick white coating except at its tip and edges which remain red. If the lingual manifestations behave characteristically the coating and subjacent epithelium will be shed from the fore backwards. At the end of six or seven days, the tongue will appear free of its coating clean red glistening and with enlarged papillae resembling a ripe strawberry or raspberry (strawberry or raspberry tongue). In nondesquamating cases, the papillae enlarge and protrude through the coating giving the tongue the appearance of a strawberry coated with cream. After the first week the tongue will take on a secondary but thinner coating.

The fauces show slight to intense congestion and at times an exudate. The exudate may be so abundant and so well organized that at first glance it may resemble diphtheria. Turbulent rhinitis, with irritation of the nostrils and upper lip may be found in the septic type. These phenomena disappear with proper treatment by the end of the first week. Desquamation commences in ten to fourteen days. The amount, severity and degree of desquamation depend on the severity and intensity of the eruption. If the eruption was mild and very faint desquamation is slight. In severe eruptive types, desquamation commences earlier and

persists longer. The parts to desquamate earliest are the regions where the eruption first appeared. Discrete, pinpoint, powdery scales may be the first evidence of desquamation on the trunk. This represents the desiccated summits of military vesicles. The hands and soles of the feet desquamate last. It is possible to make a retrospective diagnosis of scarlet fever by the appearance of the desquamation at the junction of the skin of the fingers, toes, and nails. A white line will first appear which after a day or two can be broken with a sharp instrument or by running your own fingernail through it. This separation is termed subungual cleavage. The skin can now be peeled back over the tips of the fingers, exposing smooth, purplish skin in contrast to the gray coarse desquamated skin. The finger and toe desquamation is uniform, involving all fingers and toes. The duration of the desquamation varies from a few days to six weeks or longer. Secondary or even tertiary desquamation may occur. Loss of hair after a severe attack of scarlet fever is not uncommon.

Differential Diagnosis. In the typical cases, the diagnosis is not difficult; in mild cases, with atypical symptoms, confusion with the toxic erythema of tonsillitis, measles, rubella, varicella, smallpox and drug eruptions may occur. Diphtheria requires differentiation at times.

Tonsillitis. In acute tonsillitis the erythema is nonpunctate, transient, with no desquamation. The differential diagnosis from measles and rubella has been discussed under these diseases.

Varicella and Smallpox. In the prodromal stages of these diseases, a scarlatiniform eruption may occur but it is transient and soon followed by the typical eruption of either disease.

Drug Eruptions. In these there are evidences of the disease for which the vari-

ous drugs have been used and the eruption is transient. The Schultz-Charlton extinction test is of value. The test consists in injecting intracutaneously into the area of the eruption 0.1 cc. of convalescent scarlet fever serum. In scarlet fever a complete blanching of the eruption will occur within four to six hours and persist until the eruption disappears elsewhere. To obtain the serum, persons immune to scarlet fever can be used (Dick). Scarlet fever antitoxin may be used for the test, but the objection to its use is that the patient tested may be sensitive to horse serum and react with redness in the area injected.

Diphtheria. In diphtheria, the membrane is not surrounded by a bright intense redness. There is more edema of the surrounding tissue, no palatal eruption, and no tongue evidences of scarlet fever. Smears and cultures for the Klebs-Loeffler bacillus may be required. However the appearance of the eruption after a twenty-four to forty-eight hour period of invasion will clarify the diagnosis.

Prognosis. Scarlet fever at present, is a mild disease in most cases. The most serious complications are nephritis and otitis media. Cases that begin in the most benign manner may develop a severe nephritis. This complication usually comes on late in the disease about the third or fourth week. A favorable prognosis, therefore, even in mild cases, should be made with reservations.

Susceptibility is determined by means of the Dick test read twenty-four hours after it is performed. Those developing a red areola 1 cm. in any direction at the site of the injection should be given preventative treatment. The following preparations may be used for passive immunization: *Scarlatinal streptococcus antitoxin*, convalescent serum, or scarlatinal lyophilic serum. Convalescent serum

or lyophilic serum is preferable and is given in 30-cc. doses intramuscularly. The antitoxin is used in emergencies in 2000-unit doses intramuscularly. The protection lasts for two to three weeks.

Active immunization should not be undertaken until after an exposed person has passed through the maximum incubation period. Unexposed susceptible individuals may be immunized at any time. *Scarlet fever streptococcus toxoid* is given subcutaneously at weekly intervals in graduated doses of 650, 3000, 10,000, 30,000 and 100,000 skin test doses respectively. Local and general reactions may follow. The general reactions may be slight or severe, any or all of the following: fever, headache, nausea, vomiting, abdominal pains, diarrhea, joint pains, and rashes. These can be decreased by the following precautionary measures (Melnick, *Prevention of Scarlet Fever Arch. of Pediat.*, 50:138, 1933): (1) Giving milk of magnesia on the night before day of injection, (2) restricting diet on day of injection to stewed fruit, fruit juices, cereal, tea and toast, avoiding milk and milk products; (3) restricting activity after injection, and (4) giving the injection in the late afternoon or evening and having patient retire early. Immediate general reactions are often prevented by adding adrenalin chloride (1:1000) 0.2 cc. in same syringe as the toxin. Repeat should be performed two weeks after the last dose of toxin, if positive; the fifth dose of toxin is repeated. In our experience, the three-injection treatment with scarlet fever toxoid or toxoid and precipitate immunizes only about 60 per cent of the patients as based upon the Dick test, with a 17 per cent reversal to a Dick positive in a period of nine months. Exposed children may be given sulfadiazine (0.5 gm. t.i.d.) for three to four days as a preventive.

Treatment The patient should be kept in bed for at least three weeks even in the mildest cases. *Daily warm baths* no chilling of the body *daily bowel evacuations* and *frequent examination of the urine* are indicated.

Specific Therapy *Scarlet fever antitoxin* even in the mildest case, is advisable. It helps the disease and reduces the severity and frequency of complications. The earlier the antitoxin or serum is given the more dramatic is the response. A dose of 6000 antitoxin units is sufficient in mild cases with larger doses in severe cases. With adequate antitoxin, there is rapid improvement shown by the fall in temperature and decrease in the intensity of the eruption and its rapid disappearance. Antitoxin is given intramuscularly and should be preceded by a skin sensitization test. If the test is positive and the case a mild one antitoxin should be omitted. If the test is positive and the attack severe either desensitize the patient and give the antitoxin or give *convalescent* or *lyophilic serum* if obtainable.

Of a group of fifty six scarlet fever contacts with positive throat cultures of beta hemolytic streptococci forty six were treated with 20 000 units of *penicillin* every three hours to a total dosage of 140 000 units and ten were given one intramuscular injection of 200 000 units in oil. All but two had negative throat cultures twenty four hours after treatment. The therapeutic status of *penicillin* and of the *sulfonamides* in this disease is still under study although both are definitely indicated in the complications. If *penicillin* is used early the dose suggested is 25 000 units intramuscularly every three hours for five days or until the patient has recovered from all pyogenic complications (Hirsh *et al*). They consider the value of *penicillin* in the

treatment of scarlet fever as established and "more effective than antitoxin in the prevention of complications, in reducing the number of carriers and equally as effective in decreasing toxicity."

Smallpox

SYNONYM: *Varicella*.

Definition An acute infectious, febrile disease characterized by severe constitutional symptoms and a macular eruption which appears about the fourth day and rapidly changes through successive stages to papules, vesicles, pustules, and crusts.

Incubation Period The incubation period is usually about twelve days. As a rule, no symptoms are present during this stage.

Prodromal Stage The prodromal stage is characterized by the sudden onset of fever, backache and headache more or less severe constitutional disturbances, and fleeting eruptions. The temperature on the first day may rise to 102.5 F (39 C) or 104 F (40 C) or higher and remain high for two or three days with slight morning remissions. The pulse is correspondingly accelerated and is often soft and diastolic. The respirations are rapid and labored, suggesting the onset of a pulmonary disease but without any accountable signs in the chest. Prostration is often extreme, being out of all proportion to the length of the illness. The lips and tongue are parched and dry. The skin is hot and dry during the fever but at times profuse sweating may occur.

The backache, which is the most characteristic symptom of the prodromal stage is usually localized in the lower lumbar region and less frequently in the dorsolumbar region but may become generalized extending up to the occiput and

radiating down into the legs. As a rule, it is more pronounced in adults than in children.

The headache, which ordinarily accompanies the pain in the back, may be diffuse, but is usually frontal. It may be the earliest evidence of illness. Its degree of severity varies according to the case, but is sometimes as intense as that found in tuberculous meningitis.

Constitutional disturbance is manifested by restlessness, insomnia, and delirium. Children, on the contrary are

It has an irregular distribution, generalized at times and limited to certain regions of the body at other times. Its ephemeral character is a distinguishing feature.

The scarlatiniform type may involve a large part of the cutaneous surface, but is more apt to affect certain areas, especially the extensor surfaces and face.

The petechial lesions are principally found in the abdominofemoral triangle, but not infrequently are seen in the axillae and flanks.

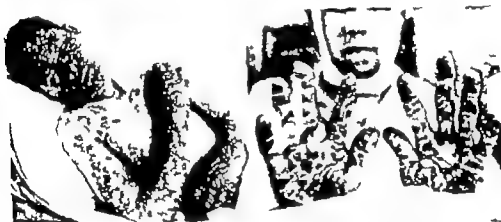


Fig. 255 *Varicella*. Left: Pocka. Right: Desquamation. (Courtesy of Dr. A. C. LaBoccetta.)

sometimes drowsy and sleepy. The severity of these symptoms is no guide to the subsequent character of the attack. Retching and vomiting are frequent. The prodromal stage usually lasts three days, rarely less. As a rule, the shorter the prodromal stage the severer the attack.

Prodromal Eruptions These usually develop on the second day of the fever and disappear generally in twenty-four to forty-eight hours. The frequency of the prodromal rashes varies in different epidemics. The lesions are petechial or purpuric erythematous, and of scarlatiniform or morbilliform character. The morbilliform is the more common type.

Eruptive Stage In most patients, the characteristic eruption of smallpox appears on the third day of the disease in the form of minute, round or oval, pale red macules. In the course of a few hours, the lesions, which become distinctly papular, increase in number and size and, on palpation, have a peculiar shotty sensation. On the third day of the eruption and the sixth or seventh day of the disease, the papules begin to assume a vesicular character. The serous contents gradually increase in amount within the next twenty-four hours. The vesicle, when fully developed, often presents a more or less marked surface depression

(umbilication) On close examination some vesicles will appear to be made up of compartments divided by vertical septa very much like the divisions of an orange. This accounts for the multilocular character of the vesicle and explains the inability to evacuate its contents completely by a single puncture. The contents which are at first clear become turbid by the ninth day of the eruption finally purulent and by the twelfth day tense pustules are formed.

On the face and scalp where the eruption is apt to be profuse the redness and swelling may be so extreme as to render the features of the patient completely unrecognizable. The eyelids as a result of the edema may become enormously puffed.

The eruption first appears on the face, scalp and wrists, then involves skin of the chest and back, later the abdomen and finally the lower extremities, the ankles and feet being affected last of all. Thus the earlier facial lesions are in a later stage of development than those on the chest and feet or other areas.

During the stage of pustulation the lesions which exhibited umbilication become distended and globular thus, effacing the central depression. The distribution of the eruption is centrifugal. The lesions on the trunk and lower extremities have little tendency to confluence but the degree of confluence may be marked on the hands and feet.

The eruption always tends to be particularly abundant on those parts of the skin which have been subjected to chemical, mechanical or other irritation. The mucous membrane may be affected. The extent of the exanthem on the mucous membranes is related to the severity of the eruption on the skin. The lesions on the mucous membranes do not pass through the stage of papule, vesicle and

pustule. As the eruption on the skin becomes vesicular and then pustular the lesions in the mouth assume a whitish or grayish appearance, with epithelial desquamation and erosion and a tendency to ulceration. The lesions are most numerous and pronounced in the buccal cavity and fauces, but other mucous membranes are liable to involvement, *i.e.*, the conjunctiva and mucosa of the nose, larynx, trachea, bronchi, vulva, vagina, and anus. The urethra, apart from the urea, usually escapes. With the appearance of the eruption, the temperature tends to fall and there is usually marked amelioration in the constitutional disturbances. However in the vesicular stage the temperature rises again, reaching its height in the pustular stage and remaining at the same level for two or more days. It then slowly falls, except in severe confluent cases or in the presence of complication, when it may persist for two or three weeks.

Period of Retrogression The first evidence of retrogression of the eruption is noted in the subsidence of the inflammatory swelling of the skin in the immediate vicinity of the pustules. This first occurs on the face where the redness and edema were most conspicuous. The eyelids become less swollen, permitting the patient to open his eyes again.

Desiccation of the lesions usually commences in those which have been the first to appear. The crusts, which are at first firmly adherent, gradually become loosened. By about the thirtieth day the separation is usually complete. The crusts persist longest in the thick skin of the palms and soles. Here they are imbedded in the thick horny layer where they are known as "seeds," and often have to be dug out with a needle, causing small excavations in the skin. If undisturbed, they may remain for weeks.

Itching varying in intensity from light annoyance to insupportable distress, makes its appearance during the stage of desiccation. Upon the completion of spontaneous shedding of the crusts, small, irregular reddish, depressed cicatrices remain. The degree of scarring varies, but is usually most marked on the face. After the lapse of three or four months, the pigmented scars assume a whitish color. Loss of hair especially after severe cases, is common but regrowth usually occurs, except in areas where the hair papillae have been destroyed.

The stage of desiccation usually begins about the eleventh or twelfth day of the eruption. In mild cases, it commences earlier in severe forms of the disease, it is delayed. Subsidence of the skin symptoms is accompanied by an improvement in the general condition, as shown by fall of temperature, disappearance of restlessness and insomnia, return of appetite and gain in weight.

Varieties of Smallpox. Three principal varieties of smallpox (discrete, confluent, and hemorrhagic) are generally described.

Discrete Smallpox. The characteristic features of this variety are the intervals of healthy skin between the lesions on the face and the attenuation or complete disappearance of the constitutional disturbances as the eruption appears. While the prodromal symptoms may vary they are usually not severe. This type of smallpox forms the great bulk of cases in an epidemic because of the partial immunity possessed by many as the result of vaccination many years before their attack.

Confluent Smallpox. This is applied to those cases in which the lesions on the face are confluent irrespective of the character of the eruption on the rest of the skin. The symptoms in the prodromal stage are uniformly severe and there is

much less remission of the temperature and constitutional disturbances on the appearance of the eruption than in the discrete cases. In most cases, the eruption is not confluent until the stage of suppuration but in the severest form, the rash is already confluent in the papular stage. The papules are thickly set. During the vesicular stage, which is often earlier than usual, considerable subcutaneous edema occurs, causing, on the face such deformity of the features that the patient soon becomes unrecognizable. The hands and feet may be swollen and exquisitely tender and painful. Upon full pustulation, lesions are likely to coalesce and form large, flat blebs, filled with purulent matter. The eruption may be confluent on the buccal mucosa, pharynx, and nares. Salivation, dysphagia, hoarseness, and aphonia may appear. Edema of the glottis may occur in very severe types.

With the beginning of desiccation, the edema subsides. Because of the depth of the lesions, many crusts may form, ultimately resulting in deeper and very conspicuous scars. Recovery is apt to be retarded by numerous complications, such as subcutaneous abscesses, boils, or tonsillar abscesses. In the fatal cases, the evolution of the eruption is very slow and there is profound prostration.

Hemorrhagic Smallpox. Two important varieties are observed.

VARIOLA PUSTULOSA HÆMORRHAGICA. In this variety the onset is unusually severe, and the subsequent course of the disease depends upon whether the hemorrhages occur in the papular, vesicular or pustular stages. The hemorrhages may be confined to one area or are generalized. Petechiae and ecchymoses may appear between the lesions or at the bases of the papules or vesicles, accompanied by hemorrhages from the mucous mem-

brane (epistaxis, hematuria, metrorrhagia and hemoptysis). Recovery rarely occurs.

VARICELLA VARIOLOSA. The incubation is usually seven to eight days. The onset is extremely violent and collapse is a prominent feature. Headache and backache are usually very intense. Vomiting is very distressing and persistent. In this form hemorrhages may appear in the skin and at the same time occur from the mucous membranes. The disease may run a fulminating course, death occurring within twenty-four hours. In most cases, death is due to cardiac failure or edema of the lungs.

Modified Smallpox or Varioloid. This is the term applied to those cases in which, as the result of a natural immunity or much more frequently of an immunity conferred by vaccination or a previous attack of smallpox, the eruption differs from that of natural smallpox. This difference is manifested by an abortive evolution of the characteristic pox. The vesicles and pustules are smaller or more superficial than usual and sometimes suppuration does not take place at all. In some cases, the lesions do not advance beyond the papular stage. Modification of the lesions occurs, whether the rash is profuse or scanty.

The constitutional disturbances are less severe than in the ordinary type of smallpox. The temperature rises to a variable height in the prodromal stage, usually drops suddenly to normal or subnormal upon the appearance of the eruption and remains so unless complications appear. Complications are exceptional and complete recovery nearly always takes place.

Complications. Among the usual complications are cutaneous abscesses, erysipelas, furunculosis, and occasionally gangrene of the skin. Eye complications,

otitis media and bronchopneumonia are observed.

Differential Diagnosis. Typical cases of smallpox are ordinarily easy to diagnose. It is in the mild and modified cases that present difficulties. In all doubtful cases, an inquiry as to exposure to infection and vaccination is important. In the absence of recent successful vaccination, the operation should be performed at once; a successful take will exclude smallpox. At times smallpox must be differentiated from varicella, pustular impetigo, drug rashes and impetigo contagiosa. In the prodromal stage, before the true eruption appears, scarlet fever, measles and typhus fever may have to be considered. The transient behavior of the eruption, simulating the latter diseases, and the development of a new eruption, accompanied by subsidence of temperature, excludes these diseases.

In varicella a prodromal stage is generally absent; if present it is of very short duration. The lesions rapidly change to vesicles in crops and are located chiefly over the covered parts of the body. They are unilocular, soft and easily broken upon gentle pressure.

Laboratory tests consist of the film test of Van Rooyen and Illingworth, the Paul test, and the chicken membrane test.

Pustular syphilids are, at times, very difficult to distinguish from smallpox. Milder constitutional initial symptoms, followed by rapid evolution of the lesions and absence of umbilication suggests syphilis. The history, presence of a chancre, darkfield studies, and serological tests for syphilis are confirmatory.

In impetigo contagiosa there is no prodromal stage, and the lesions are superficial, bullous, flat and dry with crusts which are easily removed.

Prognosis. The prognosis in smallpox depends mainly on the character and

distribution of the eruption, the nature of the epidemic, and the person attacked. In recent years, the type of infection has been mild, and the mortality rate has been very low in the United States.

Treatment. There is no well-recognized specific treatment for smallpox. Previously there was no drug available that had any influence upon the toxemia or systemic manifestations. In a report of an epidemic at a British general hospital in India, encouraging results were obtained by the use of large doses of *salicylic acid*. The effect was dramatic in many instances. Given early in the disease the vesicles, instead of becoming tense and convex, collapsed as their contents were absorbed, leaving empty shells. As a result there was no pitting in these cases and scarring was minimal. It also hastened recovery of those with second *ary infections*.

Frequent warm antiseptic baths (potassium permanganate or Dakin's solution) will relieve the severe itching and burning, arrest suppuration, prevent deep *scarring*, and hasten convalescence.

Special attention should be given to the eyes. Warm boracic acid solution washes two or three times daily will prevent accumulation of secretions, and application of petrolatum to the lids at night will prevent them from sticking together.

Vaccination. Of all the measures employed to prevent smallpox, none is so important and efficacious as vaccination with cowpox, as described by Jenner in 1790. The date at which primary vaccination should be performed varies according to circumstances. Except in epidemic regions, where vaccination should be carried out as soon as possible, the operation need not be performed until the child is three or four months of age. In the presence of any extensive condi-

tion, such as eczema or impetigo, vaccination is best postponed until the skin is normal. Vaccination should be postponed in malnutrition and respiratory infections, in the presence of a communicable disease in the immediate environment, or incomplete recovery from a recent illness. It is preferable to vaccinate in the spring or fall.

In the presence of a smallpox epidemic vaccination ought not be delayed for any reason. Except in cases of extensive eczema, an intradermal tissue culture, virus injection can be used with safety.

Technique. The site most usually chosen for vaccination is the outer site of the arm, near the insertion of the deltoid. In female infants, the outer side of the leg, a little below the knee, is sometimes chosen as the site. Before applying the lymph, the site for vaccination should be washed with soap and water followed with alcohol and permitted to dry thoroughly. The method of choice is the multiple pressure method advised by the United States Public Health Service. The under side of the arm is grasped by the operator's left hand in order to stretch the skin where the virus has been applied. The tension is continued while the virus is being inserted. A needle held firmly and parallel to the arm is pressed up and down quickly and vigorously through the drop of virus, about thirty times within five seconds in an area about $\frac{1}{8}$ inch in diameter. The needle is lifted clear of the skin each time. Immediately after the pressures have been made, the remaining virus may be wiped off with sterile gauze.

Symptomatology of a Primary "Take." Nothing is noted until the fifth day when a papule forms at the site of the vaccination. It gradually increases in size and extent. A vesicle soon develops with marked infiltration of the neighboring tissue and the formation of a red areola

A central depression or umbilication appears in the vesicle on the sixth day. By the end of the seventh day when the umbilication of the vesicle and the surrounding infiltration and areola have become pronounced the contents of the vesicle become purulent. The pustule rarely exceeds $\frac{1}{4}$ inch in diameter but the area of erythema and induration may

eighteenth day the crust falls off leaving a pigmented scar which in the course of time becomes pale and pitted.

The constitutional disturbances, in the form of fever and malaise, are usually more marked in adults than in children. In the very young the process is often entirely afebrile and glandular swelling may be absent. In some cases, the ex-



Fig. 236: Accidental Vaccinia. *Left:* Of chin and tongue. A sister had been recently vaccinated and, in some way, the child's chin and mouth had come in contact with the vesicle lesion. *Right:* Of right eyelid, plus two additional pustular lesions on the right side temporal region.

extend for a considerable distance beyond it. Small additional vesicles may occur at the periphery of the indurated area. During the next two days, the pustule and the adjacent erythema are at their fullest development. There is usually some enlargement and tenderness of the draining axillary or inguinal glands.

About the tenth or eleventh day all local disturbances begin to subside. The pustule dries to a brownish crust, the erythema disappears and the induration and adenitis subside. On the sixteenth to

seventeenth day a "beginning take" does not appear until seven, ten or fourteen days after vaccination. The longer the incubation period, the greater is the susceptibility of the vaccinated individual and the more severe the reaction. The small reaction which occurs within the first twenty-four to forty-eight hours and disappears rapidly indicates a high degree of immunity. Another type of reaction occurs between the third and fifth days when a vesicle and considerable erythema develops. At the same time, slight fever

malaise, and vague pains, with enlargement and tenderness of the adjacent lymphatic glands, appear. This reaction is termed the accelerated type or vaccineoid. Vaccination should be repeated on an average of every five to seven years, unless there is an epidemic or possible exposure to smallpox, when it should be performed at once.

Treatment. No dressing, shield or cover need be applied and instructions should be given that no cover is to be applied at any time unless the vaccination

Postvaccinal Eruptions and Accidental Vaccinia. These may be classified (Parschen's Modified) as follows:

- I. Eruptions due to contact infection with vaccine virus.
 - A In those recently vaccinated.
 - 1 Secondary infection close to original site and due to spread of the virus through the lymphatics
 - 2 Secondary accidental inoculation at distant sites on same person
 - B In those not recently vaccinated.
 - 1 Unilateral eruption on normal skin
 - 2 Accidental vaccination of diseased skin or eczema vaccination (see below)



Fig. 257 Eczema Vaccination. A sister had recently been vaccinated.

is exposed to contamination or irritation as a result of the patient's occupation. Dryness and a free flow of air should be maintained. With the development of a take the frequent application of alcohol with a moist, but not saturated, cotton sponge to the rim of erythema surrounding the inoculation will lessen the local inflammation, decrease tendency to infection, and hasten recovery. Care should be taken not to touch the lesion proper where moisture or pressure are likely to break the tender tense pustule. If by reason of discharge of lymph, crusting occurs then a large two-layer gauze dressing to be applied loosely changed daily and fastened by adhesive far outside the indurated area.

- II Eruptions presumably on heretofore normal skin.
 - A Eruptions directly following vaccination
 - B Those exanthems occurring at the time of pustulation. These are scarlatiniform, rose-billiform, urticarial, papular and purpuric.
 - C True generalized varioloid
- III Eruptions coincident with vaccination, such as pemphigus, dermatitis herpetiformis, and scleroderma.
- IV Eruptions due to mixed inoculation or infection of the vaccination site such as tuberculosis, syphilis, impetigo, etc., superimposed.

Eczema Vaccinatum (Platon) This condition is due to multiple, successive direct, autoinoculation (perhaps blood borne) of eczematous areas by vaccine virus. History of contact with recently vaccinated people is generally elicited, or

the patient has been recently vaccinated. If seropus from an early lesion is inoculated into a rabbit cornea a keratitis results (*Paul Test*). Biopsy specimens from the patient and the rabbit cornea show acidophilic cytoplasmic but not nuclear inclusions (Guarnieri bodies).

SYMPTOM This complication of infantile eczema and occasionally adult neurodermatitis, is characterized by the sudden development of discrete grouped and confluent firm umbilicated vesicopustules. The lesions develop over a period of four to seven days and are largely located on the eczematized areas. They are accompanied by fever, anorexia

and general malaise and, locally by considerable edema and pain.

The lesions dry to crusts which soon fall off leaving pigmented marks and some typical pitted scars. The infection may be severe enough to cause death by sepsis.

DIAGNOSIS It is generally easy if the above sequence of events is remembered. Also see Kaposi varicelliform eruption (p 428) from which a clinical differentiation is difficult.

TREATMENT This is largely empirical. Apply soothing applications and supportive therapy where indicated.

Sulfonamides are beneficial.

FAVUS

SYNONYMS *Thous favosa, porrigo favosa, dermatomycosis favosa, crusted ringworm, honeycomb ringworm, porrigo scutula, porrigo lupinosa.*

Favus is caused by fungi belonging to the genus *Achorion*. It usually attacks the scalp; however the glabrous skin, nails, and in rare instances the mucous membranes, may become involved.

Varieties The principal varieties are *thous tonsurans favosa*, *dermatomycosis favosa*, and *onychomycosis favosa*. *Favus dermatomyces* (favids) a generalized eruption, infrequently occurs and resembles the microsporids and trichophytids.

Incidence Favus is a disease of the poor and undernourished. It is found almost exclusively among the Russian, Polish, and Jewish immigrants.

Etiology The *Achorion schoeleinii* is the cause of most cases of favus, especially those of the scalp and nails. The group of favus parasites also includes *Achorion quinckeanum*, *Achorion gypsum*, *Achorion gallinarum*. The latter group attacks the glabrous skin rather than the scalp and nails.

Pathology The fungus does not penetrate deeper than the horny layer hence there is little if any morbid alteration of either the epithelium or the cutis. The disease begins by *favus invasionis* and proliferation within the horny layer.

The scutulum stage offers the classical picture of favus as it is usually seen. The skin surface appears sunken throughout the whole extent of the scutulum which occupies the entire horny layer. The epithelium consists of a few rows of atrophic cells. The papillae are practically obliterated. The scutulum itself consists of mycelial threads and spores, the center of which is composed of finely pow-

dered degenerated spores, while on the edges mycelial threads predominate.

In long-standing cases, the epithelium has been entirely destroyed under the scutulum and it rests only on the corium. The process usually begins in the orifice of the hair follicle and the hair is invaded and destroyed.

Symptoms The characteristic lesions are sulfur-yellow or saffron, pinhead to pea sized, cup-shaped crusts, called scutula, each of which is usually pierced by a hair. The scutula are often imperfectly formed, and appear as scattered grayish-yellow specks, or in thick yellow clusters; or through coalescence as extensive thick, dirty yellow adherent crusts. They have a mouldy odor which is fairly distinctive.

On the glabrous skin, the patches form thickly crusted, yellowish disks, which through fusion and peripheral extension may cover large areas. The lesions are frequently multiple and even numerous, and they have a tendency to simulate ringworm, forming rings which, however may be surrounded by an inflammatory halo. The lesions are more often on the face and extremities. Generalized favus is rare; an occasional case has been reported in which death occurred and infection of the stomach and intestines was found on autopsy.

When the nails are affected, they become brittle, discolored irregularly thickened, and under the free margin there are collections of crusts. No true scutula are produced. Clinically the appearance is generally not distinguish-

able from other forms of onychomycosis. The fingers are more often affected than the toes.

When the scalp is involved the hairs in the patches become dry, lusterless, wiry and loose and about their bases are convex yellow scutula. As the disease progresses alopecia and scarring supervene, often associated with sup-



Fig. 25B: Favus of the scalp (A horizon quinckeoni in 1900-01)

puration. The scars are as a rule white and depressed and similar to those which occur in papulonecrotic tuberculid. The atrophy causes a smooth, glossy, thin, paper-white, hairless patch in which the follicular orifices are absent. These patches are generally irregular in shape, with a tendency to be more or less oval, and they may be small or large and of any number.

Diagnosis. The clinical appearance of favus often simulates trichophytosis, seborrheic dermatitis, psoriasis, lupus erythematosus or folliculitis decalvans or staphylococcal dermatitis associated with pediculosis capiti. Its differentiation from ringworm and seborrheic dermatitis is established by microscopic examina-

tion or culture. In staphylococcal infections associated with pediculosis capiti, there is intense itching, pyoderma, impetigo and furunculosis.

Prognosis. Favus of the scalp is resistant to treatment and is extremely chronic. It invariably results in hair loss and a moderate amount of scarring.

Prophylaxis. Contact with domestic animals should be avoided. The use of other than one's own cap, hat, comb, and brush is a necessary precaution.

Treatment. Favus of the body responds readily to antiparasitic remedies such as are used in the treatment of ringworm. The chief ingredients are sulfur and salicylic acid or iodine. Treatment of favus of the nails follows the methods described for other forms of onychomycosis.

Favus of the scalp is treated in the same manner as resistant types of ringworm of the scalp with the exertion of extraordinary care, however, because the disease is much more difficult to cure. Treatment by medicinal means is unsuccessful and x-ray epilation is the most satisfactory method, although failures by this method are not infrequent. The technic is similar to that described for tinea tonsurans, except that thick crusts should be removed by a soap poultice before irradiation is applied, as they would otherwise absorb a considerable fraction of the dose. The epilation must be complete. In ringworm of the scalp, cures often follow partial epilation but in favus, recurrences always occur under such a condition. Even the smallest tufts of hair must be epilated manually if necessary. Strong sulfur ointment (20 per cent) must be applied immediately when the hair has fallen and must be continued daily so that all clinical and microscopic evidences of the disease disappear before the hair re-

grows. As larva is likely to cause atrophy and permanent baldness, the physician should acquaint the parents of the patient with these possibilities and should urge x ray treatment at once to a owl them as much as possible.

After the diseased hair is epilated, the following medication is helpful.

Solutions of astringents such as *Aloours* and *Vlemineck's* (solution of sulfated lime) are excellent for their detergent, antiseptic, and softening actions. The lotions are sponged over involved areas after eliminating crusts. Zinc oleate ointment is preferred by some

clinicians. The *oleates* of mercury and copper are satisfactory. The following two ointments are useful.

I	
Ammoniated mercury	10 per cent
Salicylic acid	5 per cent
Goose grease q. s.	
II	
Iodine crystals	2 per cent
Thymol	1 per cent
Oil cinnamon	1 per cent
1 g. asphor q. s.	

Resorcin ointment, a 10 per cent boric acid ointment, chrysarobin, thymol iodide, and eutophen are other efficient remedies.

FIBROUS NODULES OF THE SKIN

There are several varieties of fibrous cutaneous nodular formations (fibromas) which appear to develop in the course of a number of diseases and in others under unknown conditions. These varieties are juxta-articular nodules (*Lata Jeanseine*), disseminated nodules, *Scleroderma durum* and knuckle pads.

Juxta-Articular Nodules

These are found in chronic yaws and chronic syphilis, most commonly in the region of joints or pressure points, but occasionally elsewhere, as on the forehead, ears, and hands. They are particularly common around ankles, elbows, and knees. They are indolent, vary in size and may or may not be easily movable under the skin, which is usually normal. They are apt to be multiple, round, or egg-shaped. Trauma appears to be the exciting factor. They disappear under antisyphilitic therapy.

Disseminated Nodules

Subcutaneous nodular formations have been observed in rheumatoid arthritis, in *acromdermatitis chronica atrophicans*,

in *scleroderma* and in *panniculitis*. They must not be confused with the non-fibrous nodules observed in calcinosis, xanthomas (in tendons and tendon sheath), amyloidosis, and circumscribed myxedema. According to Swetser and Winer about 80 per cent of patients with rheumatoid arthritis show firm, painless, discrete, subcutaneous, variously sized nodules, especially over the elbow and dorsal surfaces of forearms, hands, knees, scalp, and sacral area. They develop slowly and persist for a number of years. The subcutaneous nodules (tophi) of gout are commonly located on the ear but may be present over the olecranon, fingers, hands, feet, and legs. Ear nodules (usually at tip of helix of auricle or *Woolner's tip*) in the presence of a polyarthritis are highly suspect. A certain diagnosis can be made by demonstrating under the microscope, the needle-shaped crystals of sodium urate or by a positive *microscopic* test.

These nodules must be differentiated from *hypodermic* and *disseminated* *acrocystic* erythema nodosum (nonulcerated type of *Bazin*) *gummas* in general,

and those *nodules* which appear occasionally in the course of rheumatic fever and certain other infectious diseases. Nodules from these latter sources likewise appear on the elbows, knees, wrists, and shoulders. They are intradermic or subdermic pale pink, firm and tender. They last for only several days.



Fig. 259. Juxta Articular Nodules (Jatz Jeannel) or Fibroid Gummata. Over both olecranon processes; complete resolution under anti syphilitic therapy.

Periarteritis Nodosa

This inflammatory disease of the smaller arteries and arterioles, with associated general malaise, irregular fever and multiple systemic disturbances is accompanied by skin changes (macules, petechiae, ecchymosis, lividoreticularia, and nodules) in about 25 per cent of the cases. According to Kerton and Bernstein, nodules are generally recognized as the most characteristic pathologic evidence of the disease. The nodules are transient and occur in the skin and subcutaneous tissues. They vary in size, appear singly or in crops (usually along the course of a superficial artery) and are rarely painful.

Fibroma Durum

SYNONYMS: *Fibroma simplex* (Unna), *subepidermal nodular fibrosis* (Michelson), *dermatofibroma lentiginosum* (Schreus), *noduli cutanei* (Arnig and Lewandowsky).

This affection is characterized by the presence of one or more variously sized nodules. They vary from red to dark brown and are hard, shotlike and painless.

Etiology. The exact cause is unknown but trauma is believed to play an initiating factor. The nodules may appear at any age, most often in the female and are not uncommon, although often overlooked because of their benign and painless character.

Pathology. Histologically the lesion in its early stage appears to be of an inflammatory nature. Later there are marked fibrotic changes. The result is a dense fibrosis, together with a complete loss of elastic tissue and other normal skin elements.

Symptoms. The nodules appear occasionally grow slowly and may be found anywhere on the body but usually on the legs and thighs. In all stages of the process, the lesions are smooth, sharply defined, hard and solid and movable with the skin. They are single (sometimes several are scattered) and are rarely larger than a small pea. They tend to reach a certain size, remain stationary for a variable period of time—sometimes years—and then undergo spontaneous regression, leaving a flat or slightly depressed pigmented spot. In other instances, they undergo calcareous, mucoid and xanthomatous changes. In them blood lipoids are often found increased. They do not reappear after excision. Most patients seen with this condition are interested because of cos-

metic reasons, rarely because of a fear of malignancy.

Diagnosis The diagnosis is ordinarily easy. Histologic study may be necessary to exclude xanthomas with marked fibrous changes. The histiocytoma, which cannot clinically be differentiated, is, however, histologically distinctive. According to Arnold and Tilden, histo-

Knuckle Pads

SYNONYM *Holoderma* (Förster)

Etiology Two etiological varieties are observed: essential and symptomatic. In the essential types, the cause is unknown, although in these a hereditary factor has been observed, as well as an occasional association with Dupuytren's



Fig. 240 Knuckle Pads.

cytomas are related to the xanthomas. Senior and Usher state that the cells take up iron when vital staining with colloidal iron (solution of saccharated ferric chloride) is used, and are, therefore, derived from the reticuloendothelial system. As a rule only by histological examination can the nodules be differentiated from the histiocytomas.

Treatment This is not ordinarily demanded except for cosmetic reasons. Under these circumstances, excision is the method of choice.

contracture. In the symptomatic types, trauma can be elicited as the factor and occurs in carpenters, gardeners, lumbermen, card-players, and those who bite and suck the affected knuckles.

Symptoms The lesions usually appear on the fingers over one or several proximal phalanges, rarely over the distal phalanges or thumbs. They develop slowly and tend to persist indefinitely as dime-sized nodules, which are yellowish, painless, sharply outlined, elevated, and roughly rounded. Their surfaces are

hyperkeratotic, and in winter the lesions are apt to become dried red eroded and fissured. The skin is adherent, but the lesions move freely over the underlying bone.

Diagnosis. The diagnosis is generally not difficult. The nodules must not be confused with (1) *Heberden's nodes* which are firmly attached to the bone and occur at the distal interphalangeal joints (2) *tophi of gout* in which urate crystals can be found or (3) *synovial*

lesions which are soft or give a fluctuating sensation on palpation.

Treatment. Treatment is usually not satisfactory. The application of such caustics as solid carbon dioxide, pure phenol or trichloroacetic acid may be tried applied at weekly intervals. The lesions may also be painted at twelve-day intervals with a 40 per cent suspension of salicylic acid in creol followed by the application of a protective such as elastoplast.

FISSURES

SYNONYM: *Rhagades*.

Fissures are small variously sized linear cracks which extend through the epidermis and upper derm. They are the result of injury or disease. The term "rhagades" is often confined to the radiating fissures and scars of the lip seen in infants with frank congenital syphilis.

Hangnails are in a sense similar to fissures but represent horizontal separation of the epidermis usually located just behind the nail or cuticle of one or several fingers; the separation often involves a portion of the derm.

Fissures commonly present a bright red base. Although usually dry they may be the site of a serosanguineous discharge, especially if traumatized. They are frequently tender and almost always painful. They develop readily in tissues thickened and inflamed as in all forms of eczema or dermatitis, psoriasis (especially inverted forms) and ichthyosis, particularly after the excessive use of soap in these cases. Such fissures are due to impaired local elasticity and movements and are, therefore, most often seen on the fingers, palms, soles, and flexures.

Fissures are also common on the nip-

ples of nursing women, the lip (especially the center and commissures of children) dorsum of hands in cold weather (chapping) palms of manual workers and those handling drying chemicals, around the nares in those with irritating nasal discharges, in the retroauricular region (often streptococic in origin according to Sabouraud) and the perianal areas. In diabetics, the prepuce and vulva may develop fissures.

The simple solitary fissure of the lip—usually the lower and in children—is apt to be painful, bleed easily and, if present for any length of time, present indurated borders.

Treatment. Fissures of chapping and those on the lip are benefited by frequent use of a hand lotion or emollient such as

Aqua Deet	50
Lanoli	50
Petrolat	100
Benzocaine	01

Hangnails heal rapidly if painted daily with flexible collodion.

Fissures of the nipple and perianal areas should be compressed with boric acid solution three or four times daily.

for fifteen minutes and then painted with a 25 per cent solution of silver nitrate or compound tincture of benzoin. An ointment of 5 per cent balsam of Peru is often helpful.

Retroauricular fissures are best treated by the thorough application of 1 per cent tincture of iodine succeeded by the following:

Pel Zinc Oxide	8.0
Aquaphor	32.0
Ichthammol	1.6

A chronic fissure of the lip may be helped by stretching adhesive plaster completely across the affected part to approximate and fix the edges. In some, a single suture under local anesthesia may be required for healing.

FISTULA AURIS CONGENITA

Congenital auricular fistula is generally thought to be formed as a result of imperfect or incomplete closure between the tubercle and one of the accessory tubercles which form the pinna or between two of the accessory tubercles. These fistulas are most often located on the crus helix and rarely on the lobule or antitragus. They are unilateral or bilateral malformations, often hereditary and familial. In some, only a suggestion of a fistula is present in the form of one or several shallow depressions in the skin, in others, it is a short (rarely more than 2 cm.) blind canal with a lining of epithelium, which may or may not secrete a whitish or mucoid fluid. The discharge may cause a dermatitis at the fistulous orifice. Occasionally they communicate with a small cyst which may become infected. A probe shows that the tract runs parallel to the external auditory canal, but there is no

communication with it or the tympanum.

Treatment consists in surgical obliteration.



Fig. 261 Congenital Anomaly. Note opening of fistula. Similar anomaly present in other members of family.

FOX-FORDYCE DISEASE

SYNONYMS *Lichenoid eruption of the axilla, chronic itching papular eruption of the axillae and pubes.*

Developing in connection with the poecine glands, this rare condition is manifested by a chronic, often pruritic, papular eruption.

Etiology The cause is unknown. The condition is usually observed in the

female adult. It has been considered a variety of neurodermatitis, but recent studies by Peck indicate that this disease is an endocrine disturbance in which the apocrine glands are specially involved. The histologic findings are similar to



Fig 262: Fox Fordyce Disease (Courtesy of Dr Howard Fox.)

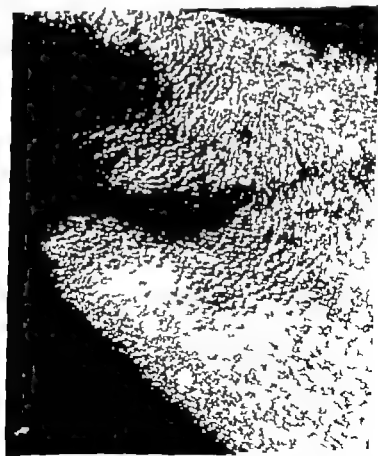


Fig 263: Fox Fordyce Disease (Courtesy of Dr C. C. Thomas.)

those found in lichenification except for the presence of marked dilatation of the apocrine glands (Fordyce)

Symptoms The disease is essentially regional, the sites of predilection are especially the axillae but may also be the pubes, labia majora, perineum, areola of the nipples, and umbilicus—all or only one of these areas being involved. Pruritus is usually present it is apt to be worse at menstruation, is often intense, but may be entirely absent. General nervousness and irritability are usual. The lesions are firm papules, sharply defined, closely grouped, smooth, and conical or hemispherical, about the size of a pinhead or larger. They are usually skin-colored, generally numerous, often glistening, and sometimes arranged in linear row along the lines of cleavage of the skin, which may have a

rugoid appearance. The surface of the papules may show hemorrhagic crusts or small centrally depressed points. Ordinarily the skin between the papules is normal or pigmented, but is not lichenified. The hair at the involved site is apt to be sparse or absent, and the papules may extend beyond the region occupied by the hair.

Prognosis The course of this affection is chronic without any tendency to retrogression.

Treatment This is usually unsatisfactory. X ray therapy has been beneficial at times. Estrogenic synthetic (diethylstilbesterol, 1 mg daily) and natural, and ovarian therapy have been found valuable in some instances. The administration of solution of potassium arsenite is occasionally followed by prompt relief of symptoms.

FURUNCULUS

SYNONYMS *Boil, furuncle.*

Furunculus is manifested as an extremely acute, painful, and tender inflammatory nodule, primarily involving the hair follicle and surrounding tissue.

Etiology Boils are caused by infection with *Staphylococcus aureus*, which invades the middle and deeper portions of the hair follicles. Characteristically the organism or its toxins cause necrosis of the follicular wall and neighboring derm. Predisposing factors in the causation of furuncles are external and internal. The external factors are irritant contact in occupations (mechanics, sugar refiners, workers with petroleum and tar products) and irritants applied for medicinal reasons. Pressure, rubbing (traded shirt collars, horseback riding) and scratching (pediculosis, scabies) are common external factors.

A special susceptibility of the patient

appears to be always necessary whether the external agents already noted are present or there are such internal factors as diabetes mellitus or chronic renal disease. In some with recurrent furunculosis, dietary indiscretions, and nervous and physical exhaustion appear to play a part. Frequently however in recurrent furunculosis the cause remains obscure. Furunculosis is not uncommon towards the termination of a severe illness, such as a generalized dermatosis, persistent miliaria, and typhoid and other fevers.

Symptoms The furuncle begins as a small, bright-red induration, which is painful from the onset. It is often centered by a hair. In other instances, it begins as a small, superficial pustule, centered by a hair beneath which redness and induration gradually develop.

Involution in this stage may occur. Usually, however, after several days the induration is the size of a pea or larger and is marked by a white central pustule which opens and discharges a little pus. Through the opening can be seen grayish yellow necrotic tissue. This in from ten to twelve days, is thrown off exposing a central cavity which fills as swelling, redness, and induration fade.

Only one furuncle may be present, but surrounding hair follicles often become infected. They are occasionally multiple and scattered here and there over the body. Furuncle of the external auditory canal is apt to be very painful; that of the upper lip causes considerable edema and occasionally thrombophlebitis, meningitis, and septicemia. A blind boil is one showing little or no tendency to point or undergo central necrosis.

Diagnosis. The sight and feel of a furuncle are so well known that few fail to recognize it on others or themselves. *Simple folliculitis* is not indurated or painful and does not develop the central slough of a boil. In *hidradenitis* of the axilla there is considerable suppuration but no necrotic central core to the rounded nodules as seen in furunculus. The *carbuncle* shows multiple points of suppuration and is larger and apt to be accompanied by constitutional symptoms. In some instances the differentiation is not at first apparent especially if perifuruncular subepidermic infections have taken place.

Treatment. *General Treatment.* This may be necessary especially in patients with repeated attacks and in furunculosis, and will depend upon the etiological indications already noted. In general, the diet should exclude alcohol, starches, seafood and cheese. In some, a diet limited to vegetables is often beneficial. Iron, arsenic and liver extract

orally are of value. A vacation at the seashore, mountains, or in the country is at times indicated. Generalized ultra-violet radiation given daily is of value. The sulfonamides are at times of benefit. *Sulfathiazole* or *sulfadiazine* (0.5 gm. [$7\frac{1}{2}$ grains] four times daily for one week) may be used.

Penicillin 20,000 units intramuscularly every three hours for twelve injections, has been successfully used in serious lesions, such as those on the upper lip and in rebellious and recurrent cases. For these patients subcutaneous injections of *staphylococcus vaccine* in an initial dose of 0.3 cc., cautiously increased as tolerated or *staphylococcus toxoid* in an initial dose of 0.1 cc., increased by same amount at five-day intervals up to a maximum dose of 1 cc. and for fifteen to twenty doses, are valuable. Occasionally 10 cc of sterile defatted milk injected intramuscularly gives dramatic results.

Local Treatment. For the single furuncle on a nonhairy area radiation with x rays (one-half threshold erythema dose daily for three days) early in its development may abort the lesion. Further local trauma by squeezing should be avoided. Several paintings with fresh tincture of iodine in the early stages occasionally help. Good results are often obtained with 10 per cent bone acid in glycine or starch spread on lint and applied to the lesion. *Penicillin* 10,000 units to 30 gm. (1 ounce) of a greaseless ointment base and constantly applied, appears beneficial and tends to protect the surrounding skin. Frequent swabbing of the surrounding areas with 70 per cent alcohol tends to prevent the development of new lesions. Good results have been obtained by the daily injection immediately around the furuncle, of a solution of penicillin and pro-

causa hydrochloride (0.1 cc. to 0.5 cc. of the mixture is slowly infiltrated) prepared by taking 1 cc. of penicillin solution (20 000 units per cc.) and drawing into the same syringe 1 cc. of 2 per cent procaine hydrochloride and slowly agitating it.

Fully developed lesions are best incised and drained following which the furuncular area and surrounding parts are thoroughly washed with a 1:1000 solution of phenacetol or mastaphen twice daily for several days and the affected area protected with sterile gauze and Carpenter's Liquid court plaster or duodhesive. Furuncles at the nasal orifices and in the external auditory canal do

best when there is applied to them several times a day *calcium penicillin* (10 000 units) or *tyrothricin* (25 mg. $\frac{3}{8}$ grain) to 30 gm. (1 ounce) of a greaseless ointment base and dry heat. Where furuncles are recurrent and in one area, such as the axilla or nape of the neck, radiation with x-rays is apparently of prophylactic value and should be given in one-fourth erythema unit weekly or in a single exposure of three-fourths erythema unit. In generalized furunculosis, especially in infants, one or two daily antiseptic baths are helpful (1 cupful of zinc sulfate or 2 gm. (30 grains) of potassium permanganate to the tub half full of hot water).

GLANDERS

SYNONYMS: *Equinia*, *farcy*

Definition An acute or chronic infection characterized in its acute form by severe systemic disturbances and often in both forms by cutaneous, nasal or oral lesions.

Etiology The disease is rare in the United States. It is due to the *Bacillus mallei* (*Malleomyces mallei*) present in the lesions and the blood. The malady is primarily an equine disease and is contracted accidentally, directly or indirectly from infected horses, as a rule, but also from mules and asses. It occurs, therefore, especially in farmers, hostlers, and veterinarians. It has been observed in laboratory workers.

The organism enters an abrasion in the skin or mucosa or is inhaled. It is an aerobe growing very easily in most media susceptible to most antiseptics and producing a severe orchitis when injected intraperitoneally into the guinea pig.

Symptoms Two varieties are described: acute and chronic.

Acute General symptoms develop in ten to fourteen days after infection in the form of pneumonic or pyemic disturbances, with chills, irregular, often high fever, headache, and muscle and joint pains. Abscesses, which break down into ulcers, form at the site of inoculation. Subcutaneous nodules (Farcy buds) or abscesses develop along streaks of lymphangitis. On the face, there may be an erysipelaslike state with edema, vesicles, bullae and gangrenous areas. When it begins in the nose or mouth nodules appear which break down into resistant, irregular ulcers with under-

mined borders. A nonumbilicated, disseminated pustular eruption simulating smallpox may appear in the course of the disease. Death occurs in ten to thirty days.

Chronic. In this the systemic disturbances are not as severe, although the



Fig. 261: Glanders. (Courtesy of Dr. R. Iph. W. Mendelson, Albuquerque, New Mexico.)

chronic form may terminate in an acute form. The usual site for the cutaneous lesion is the face, with nodules and abscesses which break down to expose nodulent, irregularly shaped ulcers with soft bases and undermined broken borders. A special form mutilating glanders involves the nasal mucosa from which there is a purulent discharge. It extends

progressively from that region and destroys the soft parts of the nose, lip, and cheeks. This form closely simulates a malignant tertiary syphilis of this area or a form of lupus vulgaris. Death occurs in 50 per cent of the cases.

Diagnosis. This is made on the patient's occupation, by culture of the organism from lesions, guinea pig inoculation, agglutination and complement fixation tests, and by the malachite green test

performed with 0.1 cc. commercial malachite green diluted 1:10,000 intradermally injected, and read in twenty-four to forty-eight hours. A positive persists longer than nonspecific reactions (Howe and Miller).

Treatment. It is symptomatic. Early lesions in the chronic form are excised or electrocoagulated. Sulfadiazine (2 gm [30 grains] q.i.d.) for three weeks has been advised (Howe and Miller).

GRANULOMA INGUINALE

SYNONYMS *Crohn ulceration, granuloma venereum, ulcerating granuloma of the pudenda, cryptogenic ulceration of the genitalia, granuloma inguinale tropicum, ulcerating granuloma of the pudenda, perforating granuloma of the thigh.*

Granuloma inguinale is a mildly contagious, chronic, autoinfectious granulomatous affection of the genitalia, perineum, and inguinal regions.

Varieties. Exuberant or vegetating, ulcerative and cicatricial forms, alone or in combination, are observed.

Incidence. The disease occurs more often in the tropics and it is endemic in the south in part of the United States. It is more common in Negroes and about twice as many males as females are affected. The majority of cases observed ranged from twenty to forty years of age. Cases have been reported from almost every country in the world.

Etiology. Granuloma inguinale is regarded as a venereal disease and the consensus is that it is acquired by coitus. Ball and Olansky obtained evidence to suggest infected public lice as transmitters.

The etiological agent of granuloma inguinale tropicum is the Donovan body, a gram-negative, nonsporulent, encapsulated bacillus. Anderson and his co-workers suggest the name *Donovania granulomatis* for it. Attempts to reproduce the disease in animals have failed; however it has been reproduced in hu-

mans by inoculation of aspirated pus from pseudobuboes of granuloma inguinale which contained no organisms other than Donovan bodies. The incubation varies from eight days to twelve weeks.

Pathology. The changes found in the tissues are those of infectious granuloma and chronic inflammation. The essential features of the histologic picture are the massiveness of the cellular reaction in which granulation tissue shows many plasma cells, the paucity of lymphocytes, the diffuse sprinkling of polymorphonuclear leukocytes, and the pathognomonic large mononuclear cells in the granulomatous tissue. These pathognomonic cells are specific for granuloma inguinale.

Donovan bodies found in this affection occur in coccoid, diplococcal, or short bacillary forms, apparently encapsulated, and they may be extra- or intracellular.

Symptoms. The disease starts as a vesicle, papule, or nodule. The surface epithelium becomes excoriated or eroded, and leaves an ulcer with a beefy-red granular base. Early lesions are often small buttonlike lesions raised above the surface or appearing as a fine granular film covering the glans penis, with occa-

GLANDERS

SYNONYMS: *Equula*, *farcy*

Definition An acute or chronic infection characterized in its acute form by severe systemic disturbances and often in both forms, by cutaneous, nasal or oral lesions

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mined borders. A nonumbilicated, disseminated, pustular eruption resembling smallpox may appear in the course of the disease. Death occurs in ten to thirty days.

Chronic. In this the systemic disturbances are not as severe, although the



Fig. 264 Glanders. (Courtesy of D. Ralph W. Mendelson, Albuquerque, New Mexico.)

chronic form may terminate in an acute form. The usual site for the cutaneous lesion is the face, with nodules and abscesses which break down to expose indolent, irregularly shaped ulcers with soft bases and undermined broken borders. A special form, mutilating glanders, involves the nasal mucosa from which there is a purulent discharge. It extends

sional nodules gently bulging the surface.

Another variety of early lesion is a raised, velvety smooth tuft of granulation tissue situated at the mucocutaneous border of the vaginal orifice, at the preputial orifice, or on the glans or inner surface of the prepuce. The margin of the lesion is sharply defined. The primary lesion in the female is usually found on the vulva or vagina. Occasionally however it is found on the cervix or in the perigenital zone. The granulation tissue if traumatized, bleeds easily. The lesions are not painful, this accounts for the fact that patients rarely seek medical care early in the course of the disease. The inguinal manifestations are secondary to the genital lesions. Occasionally inguinal lesions appear without trace of a genital lesion, although a history of a previous genital lesion which healed spontaneously is frequently obtained.

The lesions show very little tendency to heal; they spread by continuity or contiguity. Extension is often slow showing only insidious progress. Occasionally the spread is more rapid; there is a predilection for moist contact surfaces, particularly in the cruroscrotal folds. Daughter lesions frequently develop near the larger lesions. These coalesce after a time to form extensive ulcerative processes. The advancing border of the lesion has characteristic rolled edges while the granulation tissue encroaches onto the bordering epithelial surface.

The ulcerative process may remain more or less stationary for many years. Secondary pseudoepithelioid enlargement of penis, scrotum or labia may occur. The lesions may show a tendency to scar formation at one margin and chronic progression at another. Exacerbations of progression are common and sometimes quite extensive. The lesions,

when present for several months to years, have a sour smelling, peculiarly pungent, characteristic odor. Ultimately marked impairment of general health occurs and anorexia, anemia, and debility develop, ending in extreme cachexia and death.



Fig. 267. *Granuloma Venereum*. Extragenital location (smooth and neck). Note lesion on right lateral mucosa. Note lesion left side neck. The lesion on right lateral mucosa, on the level with the upper teeth, was an elevated, dark red, granular plaque, well-defined, extending back from region of Simonson duct orifice to rear molar area. It was neither tender nor painful. There was bilateral lymphadenopathy. There was likewise lesion in the groin, typical of *granuloma inguinale*.

Clinical Types. *Exuberant.* In this variety the ulcerative area is covered by a hypertrophic, vegetative granulation tissue which is soft and velvety to the touch and beefy red in appearance. The edges are rolled raised above the



Fig 265: Granuloma Inguinale. *Left:* Located on prepuce and corona of penis; nine month duration; simulated chancre so treated for three months; serology negative for syphilis. Fourteen injections of bismuth of novofulcin; rapid improvement. *Right:* Typical location.



Fig 266: Granuloma Inguinale. *Left:* In and on labia majora with six smaller lesions here and there over perineum. *Right:* After novofulcin. Complete healing but with considerable destruction.

lymphogranuloma, chancroid, or syphilis. Hence the term "pseudobubo" to describe the indurated inguinal swelling or suppurative abscess that so frequently follows a primary focus on the external genitalia in granuloma inguinale. It is not an adenitis *per se* but a subcutaneous granuloma.

parts affected include the oral cavity, lips, throat, neck, nose, chest, cheek, thorax, and liver.

Complications *Fusospirochetosis* is the most common complication of granuloma inguinale. The lesions become ulcerated, progressive, foul smelling, and painful and are often refractive to spe-



Fig. 269. *Granuloma Venereum*. *Left* Involving glans penis, scrotum, and groin. There are several isolated lesions on scrotum. *Right* Of glans penis, ulcerating chancre. Wassermann and Kahn 4 plus. Darkfield negative. Lesion failed to heal under bismuth injections, but cleared rapidly under fudin.

The sites of predilection of *granuloma inguinale* in order of their frequency are

1 External genitalia.

- (a) In the male: Glans, prepuce, shaft of penis (about inguinal lymph nodes)
- (b) In the female: Labia minora, labia majora, clitoris, vaginal orifice

2 Inguinal region above or along its primary genital lesion.

3 Perineum, anal region, buttocks.

4 Cervix uteri.

Most of the extragenital lesions are secondary to pudendal lesions, although in a few instances pure extragenital lesions have been described. Extragenital lesions occur in about 6 per cent of all cases of granuloma inguinale. The

cific therapy. Syphilis, chancroid, and lymphogranuloma venereum may be co-existent. Occasionally superimposed malignancy has been found to complicate the picture. Secondary elephantoid enlargement of the penis, scrotum, or vulva and clitoris may occur.

Diagnosis Granuloma inguinale tropicum must be differentiated from tertiary syphilis, phagedenic chancroid, and serpiginous venereal ulcer. Its location, its chronic evolution, absence of nodular infiltrations, and its symmetrical arrangement and negative serology differentiate it from syphilis. From phagedenic chancroid it may be distinguished by its extremely chronic evolution, by

surface and the margins are never undermined. The granulation tissue bleeds easily and can be curetted or scraped off leaving a shallow granular base.

Ulcerative In this type the ulcerative area is covered by a foul smelling membranous exudate. These lesions are apt to be somewhat painful. The lesions may be ulcerative from the start but

The scars are often keloid in type. Donovan bodies are found in tissue taken from the scarred zone.

It was formerly believed that granuloma inguinale was a disease of the skin and corium and not of the lymphatics. The demonstration of the pathognomonic cells of granuloma inguinale in regional lymph nodes showing mild focal

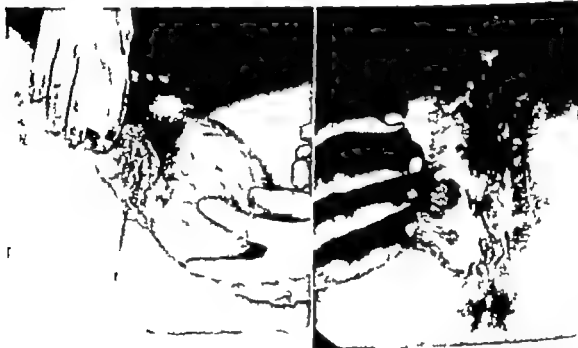


Fig. 268: Granuloma Venereum and Lymphogranuloma Venereum with Pseudo-*elephantiasis*. *Left:* Of vulva. Frei test positive. Donovan bodies found in microscopic section before vulvectomy. *Right:* Vulvectomy and electrocoagulation of remaining lesions.

usually they result from the exuberant type when it becomes secondarily infected with other organisms, particularly fusespirochetes.

Cicatrical A chronic cicatrical type of granuloma inguinale is occasionally encountered in which the granulomatous process attempts to heal itself. Where healing seems to have taken place, the area is slightly elevated or bulging and is covered by depigmented epithelium or scar tissue. Small scarred granulomatous lesions are scattered irregularly at the periphery of the progressive scar

reactions and perilymphadenitis reveals the fallacy that has been preserved in the literature as to lymphatic involvement. Automoculation may account for many such occurrences; however when an inguinal swelling precedes the typical inguinal lesion it is probable that the lymphatics played a role in the dissemination of the disease. Excised tissue from such inguinal lesions studied microscopically reveals a diffuse granulomatous reaction in the papillae, corium, and subcutaneous tissues and is not comparable to the adenitis found in venereal

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 - (1) In the male: Glans, prepuce shaft of penis (without incisional involvement)
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GRANULOMA PYOGENICUM

SYNONYMS *Septic granuloma, botryomycosis hominis, telangiectatic granuloma.*

Granuloma pyogenicum is a benign, vascular granulomatous tumor characterized by rapid growth, easily and freely bleeding on slight trauma.

Incidence The lesions occur more often during middle life and equally in both sexes.

Etiology The cause is not definitely

either bright red or brownish red. They may secrete a foul-smelling exudate. The lesion consists of very vascular granulation tissue covered by a thin layer of epidermis. The sites of predilection are the fingers, hands, scalp, lips, buccal mucosa, toes, arms, and feet. Subjective symptoms are absent.



Fig. 270 Granuloma Pyogenicum.

known. It is presumed to be due to staphylococci, probably the *Staphylococcus aureus*. Most of the cases follow trauma.

Symptoms The disease is characterized by a rapidly growing lesion, often at the site of injury. The lesions are either sessile or pedunculated and vary in size from a pea to a walnut. They are vascular and bleed easily. They are

Diagnosis Granuloma pyogenicum is distinguished from *granuloma* (proud flesh) by its firmer consistency. The lesions of *bromoderma* and *dermatitis vegetans* are multiple and larger in size. Granuloma pyogenicum is differentiated from *ulcerative hemangioma* by the fact that a hemangioma is present at birth or shortly thereafter and is softer and more compressible.

its granulomatous character and by its retracted border. The *serpiginous venereal ulcer* starting from an ulcerated chancreoid hubo may very closely resemble granuloma pudenda; the diagnosis can only be made by very careful microscopic investigation.

Prognosis. Cases of granuloma inguinale are cured when treatment is started within the first six months of the infection. Over 50 per cent of patients with lesions of several years' duration will respond, although more slowly, to treatment. Recurrences in this group are very frequent. Neglected or therapy-resistant patients may harbor the disease for ten to fifteen years. With increasing age, declining health and resistance, there is a gradual activation of the lesions. The ulcerations often occupy the whole pudendal region, lower part of the abdomen, buttocks, and thighs. The patient becomes anemic, bedridden, cachectic, and finally dies.

Treatment. The effectiveness of chemotherapy decreases in direct proportion to the chronicity of the lesions. Early granuloma inguinale responds rapidly to therapy with various *antimony* preparations. *Tartar emetic*, 1 per cent, may be given intravenously in ascending doses beginning with 1 cc and increasing the dose by 1 cc until a dose of 10 cc is reached. It is administered every two or three days. When the maximum is reached, the dosage is decreased by 1 cc in descending strength until it is reduced to the initial dosage. After a two-week rest period, medication is again started. Toxic manifestations, such as joint pains, sore gums, anorexia, nausea, and vomiting, are often encountered with tartar emetic.

Fuadin (Winthrop) has proved to be superior to tartar emetic. It is more convenient to administer and toxic re-

actions are seldom encountered. The solutions are stable and are available in 1.5-, 3- and 5-cc ampoules. The initial dosage of fuadin is 1.5 or 3 cc, intramuscularly. The dosage is increased to 5 cc and given three times each week. Where lesions remain refractory to treatment with one antimony preparation, a change to another is recommended. Usually, definite improvement may be noted in four to six weeks. To avoid recurrence, medication should be administered weekly for six months.

Some of the early cases, and particularly the chronic cases of two to three years' duration or longer, may prove resistant. If the lesions are localized, they may be excised. If the lesions are extensive, several courses of *radiation therapy* should be administered. The dosage should be similar to that employed in the treatment of skin cancer. An antimony compound should be administered along with radiation therapy and should be continued for six months after cessation of x-ray therapy.

Lesions resistant to antimony may harbor a superimposed fusospirochetal infection. Fusospirochetosis may be treated with injections of *penicillin* and by *sodium perborate dressings* or by applications of a mixture containing equal parts of *zinc peroxide* and *cod-liver oil*. Topical application of the *arsphenamines* is employed to eliminate the fusospirochetal infection. A suspension of 20 per cent *podophyllin* in olive oil applied to recalcitrant lesions for five to seven days has been recommended and should be used in conjunction with the antimony.

Kupperman, Greenblatt, and Deust have had good results with *streptomycin* given from five to forty-five days in total dosages of 5 to 50 gm. According to them, Donovan bodies disappear from lesions in two weeks and pain rapidly subsides.

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Pathology The histopathology reveals numerous newly formed blood vessels and young connective tissue with lymphocytic and plasma-cell infiltration

Prognosis The prognosis is favorable

Treatment Treatment consists of complete destruction by *electrocauterization* or *electrodesiccation* followed by dressings of 5 per cent ammoniated mercury ointment



Fig. 271: Granuloma Pyogenicum.
(Courtesy of Dr. Jacques P. Gorquiere)



Fig. 272 Granuloma Pyogenicum.

HAIR AND SCALP DISORDERS

Introduction. The hair is ectodermal in origin and consists of modified horny epithelial cells. It covers an extensive area of the skin except the palms of the hands, soles of the feet, and the dorsum of terminal phalanges. Hair is abundant in the regions of the scalp, pubis, and axillae of both sexes, as well as on the chest and face of males.

The color of hair varies in accordance with the amount of pigment (melanin) present. The number of hairs on the scalp is calculated at 1000 to the square inch or 120,000 to the adult head.

The rate of hair growth is of practical interest because it varies with age. Hair growth above the skin level is very rapid and ranges from 2 to 3 mm. every ten days and about $\frac{3}{4}$ inch per month. When the hair is longer (say 10 to 14 inches in length) its growth is slowed down to about one half the former rate ($\frac{3}{8}$ inch per month). The growth of hair is greatest in young women between the ages of sixteen and twenty-five years. A growth of 7 inches per annum has been recorded among young women. The hair grows faster during the summer months. The frequent cutting and shaving of hair does not influence its growth. The average length of hair is 22 to 28 inches. The hair rarely exceeds 36 inches in length.

Washing the Hair. Women wash their scalps once a week or once a month. Men wash their scalps each day or once a week.

Frequent washing of the scalp is not so necessary as is usually supposed, provided the hair is regularly brushed and combed. Women of tropical countries, where the hair is remarkable for its lux-

uriance, length, and gloss, very rarely wash their scalp. It is injurious to leave the hair wet. No damage occurs when the hair is adequately dried. It is necessary to cleanse the hair more frequently when it is thin and the scalp oily than when it is coarse and the scalp dry. For oily hair and scalp the following is beneficial.

Pol. yollaja	9.0
Pol. capsaica.	1.5
Acon. carb.	1.8
Aq. dest.	3.8
Perfumed spirit	12.0
Alcohol, q. s. ad	240.0
M. Macerate eight days and filter (P.R. B. III)	

Powders provide excellent shampoo materials for oily scalps with long hair. Bunched strands of hair are dusted carefully with potato starch, plain talcum powder, orris powder, or the following:

Amo. h.	50.0
Soda bicarb.	50.0
Perfume, q. s.	

The powder is then removed by repeated brushing six to twelve hours later with cotton moistened with alcohol. Tar soap is preferable as a shampoo for individuals with dry scalps. The following for dry scalp (P.R. B. III) is beneficial.

Cantharidin	0.06
Acetone	5.0
Castor oil	50.0
Alcohol, q. s. ad	100.0

Dull Hair. Dull hair is the result of insufficient brushing and combing, or of the very frequent use of the curling iron or of the "permanent wave." The hair looks invariably dull a few days after a permanent wave. This dullness is avoided by applying an oily lotion at least one hour before visiting the hairdresser.

Dry Hair Dry hair is produced by washing the scalp frequently and employing lotions containing alkalis or alcohol

Diffuse Falling of Hair

Diffuse falling of the hair occurs without any disease of the scalp. The most common causes of this type of falling hair are fevers. Sabouraud observed that diffuse falling of the hair occurred when the temperature of the body rose above 103° F (39.5° C) and that it did not occur until this temperature was reached regardless of how long a lesser temperature continued. The hair of patients running a temperature of 103° F (39.5° C) or more begins to fall from two to six weeks after the onset of fever. (See also p 71)

Loss of hair may follow surgical shock, childbirth or miscarriage. A certain amount of hair loss accompanies the menopause and old age. Debilitating diseases leading to premature senescence are accompanied by falling of the hair.

Treatment The treatment of diffuse falling hair without scalp diseases is directed to *improving the general health and increasing scalp circulation* by employing mechanical and electrical agents. *Ultraviolet light irradiation* of the body and scalp is useful and commendable.

Large doses of *riboflavin* are beneficial. A daily dose of 0.12 gm (2 grains) of *desiccated thyroid gland* should be given in every case and continued until toxic symptoms are noted. The local use of drugs for this purpose is of doubtful value.

Hirsuties

SYNONYMS: *Hypertri hosis, polytrichi, superfluus hair*

Hirsuties identifies excessive growth of hair on regions of the body where it is natural to have lanugo or downy hair.

Etiology Although it is believed normal hair formation results from inherent qualities within the hair follicle and endocrine influences. The cause of *hirsuties* cannot be determined in the majority of patients. The condition has been associated in comparatively few cases with tumors of the suprarenal cortex, pri-



Fig. 273: Hirsutism. Of chin and forearms.

tary gland pathology, thyroid gland disease, and with ovarian dysfunction. The length of time since the onset of the hypertrichosis or masculinization is of value in excluding disease or neoplasm of the ovary, adrenal and pituitary. In pituitary hypertrichosis, the x ray films may show enlarged sella turcica or perimetry may show defective visual fields. In adrenal lesions, an abdominal mass may be palpable. Arrhenoblastoma of the ovary is not unusually associated with

hypertension, disordered carbohydrate metabolism, osteoporosis, or polycythemia. A relationship between heterosexual hypertrichosis and certain instances of adrenal hyperfunction appears certain. The appearance of hypertrichosis during the menopause is probably due to altered ovarian secretion. Transitory hypertrichosis has occurred after pregnancy and after long periods of amenorrhea.

Bessell and Williams suggest hyperadrenocorticism as the basic cause in many cases.

Prognosis Cases of hirsuties resulting from endocrine disturbance have been successfully treated with glandular extracts, although internal therapy is generally unsatisfactory.

Treatment Large doses of ovarian follicular hormones and mammary extracts are occasionally helpful. There are many local methods for the destruction of superfluous hair of which the most satisfactory is electrolysis. Irradiation by x rays is contraindicated and should be avoided.

Removing the hair by a depilatory by shaving, and by pulling the hair singly with forceps or in groups by the aid of wax are merely palliative treatments. For legs and arms, the simplest and most effective of these palliatives is the electric razor.

A commonly used depilatory powder consists of

Titanium oxide	10
Boric sulphide	50
Zinc oxide	100
Pulverized q. s. ad	300

The powder is mixed in warm water to form a paste which is applied thickly to involved parts and allowed to remain from three to seven minutes. The parts are then washed, leaving them hairless. Cold cream or calamine lotion

is finally applied to prevent irritation from epilation.

The hair can be made less conspicuous by bleaching with the following mixture

	I	
Glycerol		15.0
Aqua ammoniac		4.0
Liq. hydrogenii disodii		100.0
	II	
Moonshine (Merck)		

Canities

SYNONYMS *Gray hair atrophy of the hair pigment, trichosis discolor poliothrix hirsutaria.*

The hair becomes gray to some extent in chronic toxemia. Diseases of the intestinal tract have in many instances produced canities. Hot climates, malaria, anemia, and tuberculosis have been accompanied by premature grayness. A familial tendency to premature grayness as a whole or in patches has also been reported. It has also been ascribed to prolonged mental strain, anxiety worry and other conditions leading to lowered vitality.

Acquired grayness of the hair is seen in leukoderma and alopecia areata, and it may follow shock. Casualties from shell shock have shown definite grayness of the hair within two weeks following onset of shock, a period much shorter than the time necessary for the growth of new hair.

The color of the hair has changed to yellow red, and black following severe illness and in highly emotional states. Green hair blue hair and other unusual colors are due to the local action of chemicals in hair lotions. These unusual colors may also occur among workers in copper cobalt, and indigo.

Treatment *Scalp massage* is an important therapeutic measure because it

improves the circulation in hair follicles. Only applications darken gray hair. Exposures to the *mercury vapor lamp* and to strong *sunlight* darken the hair for short periods.

The intake of *pilocarpine* and its external application have definite effects in darkening the hair.

A dye of 1 to 3 per cent solution of *silver nitrate* is perhaps the most commonly employed preparation. This is accomplished by thoroughly moistening the hair with 1 per cent *silver nitrate* and drying it in *sunlight*.

Henna and *walnut extract* are safe vegetable dyes. The least dangerous dyes contain henna preparations blended with indigo to obtain various shades ranging from light blond to deep black. The hair is defatted with soap and alkali washes before the dye is applied. Repeated hair dyeing leads to dryness and brittleness.

Dyes containing coal tar products are condemned because they frequently produce dermatitis.

Ichthyosis of the Scalp

This condition is first recognized as a rule around the age of two or three. Hair growth is poor especially over the vertex. The hair is fine, apt to be curly and the scalp is covered with a varying number of more or less adherent, brownish horny scales. When removed they expose what appears to be a perfectly normal white but dry scalp. These scales have been present since birth and the condition has gradually become more marked. The general skin surfaces, if examined are usually found to be ichthyotic.

In almost all cases of ichthyosis (see p. 414) both skin and scalp are normal at birth and ichthyosis appears several months later.

Sabouraud recommends the daily use of the following

Glycerin. anili	20 g
Ol. cadisi	10 g
Tr. soapbar, q.s. emul-sor.	

Ringed Hair

SYNONYMS: *Pili annulati*, *Leukotrichia annularis*.

Ringed hair is an extremely rare condition characterized by alternate white and pigmented bands which are narrow and ringlike. It may be hereditary and occurs in normally pigmented hair and in hair turned gray in the process of senescence.

The condition is probably due to the presence of gas filled spaces within the medulla and cortex of the hair. This makes the outer cortical cells opaque and thereby conceals its pigment.

There is no satisfactory treatment for ringed hair.

Pili Incarnati

SYNONYMS: *Ingrown hairs*, *pili recurrenti*.

Symptoms. This condition is occasionally seen involving one or two follicles on the bearded region in white males but involving many follicles of the bearded area in Negroes. It is excessively common in the Negro.

The hair apparently cut too short by close shaving of stretched skin grows through the side wall of the follicle into the derm. With further growth in this new position foreign-body irritation with swelling occurs. They heal without scarring. The type of hair in the Negro appears to make him more susceptible. In these, as Pinkus points out, the free end of the hair emerges normally but because of its strong curved tendency grows back and down into the skin. In

the Negro, a chronic papular largely noninflammatory often keloidal, sometimes pustular eruption is the result—always developing several years after shaving has been started.

It is differentiated from *sycosis ovalis* by the fact there is no interfollicular



Fig. 274 Pili incarnati. Common in Negro in whom it must not be confounded with *sycosis vulgaris*.

lar cutaneous inflammation, no central piercing of pustules by a hair and there is none of the acute reaction common to *sycosis vulgaris*.

Treatment. Treatment consists in the puncture of the inflammatory papule and the removal of the hair. Treatment in the Negro is difficult.

Atrophy of the Hair

SYNONYM *Atrophie pilorum propria*.

Atrophy of the hair is a general term identifying various atrophic changes resulting from infection by vegetable parasites. It may also occur in conjunction

with general systemic conditions which impair metabolism as in phthisis, syphilis, and diabetes.

Varieties. The commonly known forms of atrophy of the hair include fragilitas crinium, trichorrhexis nodosa, monilethrix, pedia, lepothrix, and linea nodosa.

Fragilitas Crinium. This form is characterized by extreme fragility of the hair. The hair may be split into many filaments, or it may be so brittle as to break off during brushing and combing. Too frequent shampoos, permanent waves, and excessive use of the hot curling iron may be etiologic factors.

Treatment consists of cutting the hair and massaging the scalp.

Trichorrhexis Nodosa. It is characterized by nodular enlargements of the hair shaft with consequent fracture. A brush-like expansion of the hair proper occurs at site of fracture. The mustache is the usual site of predilection, although the beard and scalp may be occasionally involved.

The disease is persistent and rebellious to treatment.

Monilethrix. A rare malformation of the hair characterized by regular fusiform swellings, often hereditary and familial.

The child is born with normal appearing scalp, hair eyelashes, and eyebrows. In about six to eight months, the scalp hair falls and does not regrow or does so poorly. At each hair follicular orifice, there develops a pinhead size, keratotic papule with reddish base, especially on the nape of the neck. The entire area gives a rasplike sensation on palpation. Hair growth, when it occurs, is irregular. The hairs vary in length but rarely measure more than 1 to 1½ inches. All are dry and the shorter ones break off close to the point of emergence from the scalp.

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Under a lens, one finds dry broken fine hair as well as the typical beaded or moniliform types, i. e. regular expanded and thinned-out areas along the length of hair. Only a few of these hairs may be present or they may be numerous, but are dry lusterless, and short.

This condition is incurable. Palliative therapy consists in the constant use once daily of equal parts of oil of cade, lanolin and petrolatum.

Piedra (Trichomycosis Nodularis Trichosporous Nodosa) Piedra is an infection characterized by the development of minute pinhead sized hard brown nodules on the hair of the moustache and beard. The nodules are very small and not unlike nits in appearance. The disease is due to a fungus known as *Trichosporon ovoides*.

Treatment consists of frequently washing the hair in a hot solution of 1:1000 bichloride of mercury.

Lepothrix (Trichomycosis Palmellina) Lepothrix is a rare infection of the hair of the axillary and genital regions characterized by studs of bullous swellings. Infection is restricted to the hair shaft. Castellani is of the opinion that lepothrix is due to a mycotic infection of the hair and that the bullae are due to "bacillary like mycelial hyphae."

Treatment consists of clipping the hair and applying a solution of bichloride of mercury (1:1000) or 10 per cent colloidal sulfur in a base of greaseless cream.

Tinea Nodosa Tinea nodosa is a fungus infection of the hair associated with seborrhea and characterized by dark brown irregularly shaped nodules upon the hair shaft. The lower portion of the hair is usually free from infection, leaving the root in good condition.

Tinea nodosa is treated by frequently shaving the hair or by clipping it short

to permit the adequate application of parasitocides.

Pediculosis

SYNONYMS *Phthiriasis, lousiness, pediculosis corporis, pediculosis vestimental.*

Pediculosis corporis, due to the body louse is associated with severe itching and the appearance of parallel scratch marks, or erosions, crusting and urticaria all of which are secondary lesions. Secondary infections and abscesses occasionally occur. Although occasionally found in hairy areas, the body pediculus commonly lives in the seams of clothing and merely feeds on the skin. Ova may be found, however on hairs. The typical pediculus bite is an erythematous spot with a pinpoint hemorrhagic center. The sites of predilection are the buttocks, abdomen, shoulder and extensor surfaces where the cutaneous surface is in close contact with clothing.

In chronic infestations, the skin may show few secondary lesions (although in characteristic locations) but may show extensive pigmentation (parasitic melanoderma).

Treatment The affection is readily eradicated by daily baths, the application of a parasitocidal ointment, and by changing the underwear. Sterilization of clothing is best accomplished in the autoclave or insufflation of D.D.T.

Pediculosis Capitis This is invasion of the scalp by the pediculus or louse. It is characterized by local itching, and the presence of insects and their nits. The nits (eggs) are millet seed sized, egg shaped shiny bodies with their smaller ends firmly cemented to the hair shaft. Unlike the particles, or scales, of "dandruff" they are not easily brushed off the hair.

The occipital area and areas behind the

ears are common sites for hair-carrying ova.

Itching of the scalp, even in adults, and especially in women, means a careful search for the pediculus or its ova.

Pediculus capitis may be complicated by cervical adenitis, eczema, impetigo, and furuncles.

Pediculus capitis is treated by daily applications of 2 per cent ammoniated mercury ointment for a period of several days to destroy living insects. The nits are resistant to soap and water but are destroyed by acetic acid diluted in water: one part acetic acid to four parts of water.

Sabouraud is of the opinion that free applications of plain petrolatum destroy the parasites by suffocating them. He adds 50 drops of *xytol* to 80 gm. of white petrolatum to destroy nits and maesta.

Female children and adults with long hair are best treated by lying on the back with the head hanging over the edge of a table; let the hair hang down into a bucket placed on the floor. A quart (1000 cc.) of an aqueous solution of carbolic acid (1:40) is poured over the head so that it may run through the hair. The hair is thoroughly dried and devitalized nits removed with a dust, or fine, comb moistened in vinegar. A 1:4000 solution of mercury bichloride may be substituted for the carbolic acid solution.

Mild cases are treated for a number of days by *trieture of delphinium*. Equal parts of kerosene and mineral oil applied for a few days at bedtime is equally effective.

DDT powder (5 per cent in talc) thoroughly applied and allowed to remain on the scalp for ten days, will kill both existing and hatching lice.

Cupress (Bierck) is specific. Three to

four tablespoonfuls is rubbed into the hair and scalp; avoid eyes. Wait one hour and then wash hair thoroughly with warm water and soap. While the hair is still damp, comb out nits and dead lice with a fine comb. *Cupress* is inflammable.

Pediculus Pubis (Crab Louse)
This parasite inhabits the hair about the genitals. The hair of the legs, chest, eye-



Fig. 275 *Pediculus Pubis*. Note numerous yellowish (on hair) and gray (on skin of left thigh) specks which are nits and lice.

lashes, eyebrows, and axillary region can be involved in *pediculus pubis*. The nits in this case become attached to the hairs very close to the skin. The maest are often overlooked because of hiding within follicular orifices of the skin.

Maculae ceruleae are pea- to larger sized, slate-colored or bluish spots, occasionally found in association with *pediculus pubis* and are believed to be due to a pigment introduced into the skin by the organism as it feeds.

The chief symptom consists of severe itching, complicated with eczema, boils,

and pustules. Pubic pruritus always requires a careful search for pubic lice.

Cutting the hair short removes a certain number of the ova.

Nightly applications of 10 per cent *calomel ointment* and an oily solution of *xylol* (1:30) are efficient treatments. The following solution is very satisfactory and pleasant.

Hydrarg. cretae.	0.1
Alcohol	120.0
Aq. menth. pip.	120.0
Sig. Apply twice each 12 hr. for four days.	

An ointment of 1 per cent *yellow oxide of mercury* is useful when the eyelashes and eyebrows are involved.

Cupress is specific. Apply the solution to the pubic hair wait ten minutes and

reapply. One hour later wash parts with warm water and soap.

Sutton advises 0.5 per cent *D.D.T.* (dichlorodiphenyl trichlorethane) in cold cream. A thin layer of the cream is rubbed into all hairy parts and a bath taken twelve hours later. Itching stops in half an hour, pediculi die and the ova do not hatch. It should be repeated every other day for ten days.

Plica Polonica

Plica polonica is a condition in which the long hair of women becomes glued and matted together to form appendages simulating a cow's tail. The condition is secondary to pediculosis capitis and is the direct result of neglect and filth.

HERPES SIMPLEX

SYNONYMS: Cold sore, fever blister, herpes febrilis.

Herpes simplex is an acute inflammatory affection characterized by the occurrence of one or several groups of vesicles resting on an erythematous base.

Varieties. The several varieties of herpes simplex include herpes facialis, herpes progenitalis, herpes menstruatus, and recurrent herpes simplex.

Incidence. Herpes has a familial tendency. Certain individuals are predisposed to it and in them the affection is often recurrent in the same locations. Andrews and Carmichael are of the opinion that 50 to 75 per cent of normal individuals are carriers of the virus of herpes simplex.

Etiology. The cause of herpes is a filterable virus. The herpes simplex virus is related to meningoencephalitis. The inoculation of a rabbit's cornea with vesicular contents of herpes simplex produces a severe keratoconjunctivitis followed by meningoencephalitis. This laboratory experiment definitely proves the presence

of the causative virus within the vesicle. The virus appears to remain in the body throughout life once infection has taken place. The disease is mildly infectious. That a large percentage of individuals are carriers of herpetic virus is shown by the occurrence of herpes simplex following an illness or minor local injury. The origin of herpetic virus is unknown.

There are many supplemental factors which precede and precipitate the appearance of herpes simplex. These include (1) indigestion, gastrointestinal disturbance, (2) exposure to sunlight, wind and inclement weather, (3) colds, rhinitis, bronchitis, etc., (4) menstrual disturbances or the appearance of normal menses; (5) sexual intercourse; (6) pneumonia, spinal meningitis, influenza, malaria, fever, therapy, vaccinations, protein infection, (7) infectious foci, teeth, tonsil, sinuses, genitalia, (8) ingestion of drugs (iodides) and certain foods, such as chocolate, cheese, nuts,

shellfish, and (9) local trauma, such as brushing of the teeth or irritation of the lips and face.

Pathology The histopathology of herpes simplex is characterized by a true coagulation necrosis in the prickle-cell layer of the epidermis. The vesicles are formed by elevation of the prickle-cell layer of the pars papillaris. Marked edema and dilation of the blood vessels occur in the corium with perivascular infiltration by leukocytes, connective tissue cells, and a few mast cells. Herpes simplex in man is one of the few virus diseases that do not lead to lasting immunity. In animals, immunity is very strong, while in man it is very doubtful whether any immunity follows an attack of herpes simplex.

Symptoms Herpes simplex is characterized by grouping of minute vesicles upon a mildly inflammatory base. The vesicles at first contain a clear exudate. Vesicular formation is followed in a few days (three to six) by desiccation and crusting which later become seroparous. Although the lips are the most frequently involved, single or multiple groups of vesicles may occur anywhere on the body.

Herpes Facialis. This occurs more often on the vermillion border of the lips about the oral commissure (herpes labialis). It also occurs on the nose (herpes nasalis), the cheeks, and the auricles. The mucous membrane of the mouth, the throat, and the conjunctivae is also occasionally affected. Herpetic lesions of the mucous membrane of the mouth and pharynx rapidly undergo maceration and appear as whitish round or oval erosions surrounded by a narrow red areola and are known as canker sores. Mild constitutional symptoms usually accompany herpetic lesions of the mucous membrane.

The eruption is usually preceded by a sensation of tingling or burning. Slight systemic disturbances, such as malaise and headache, are usually present in the ordinary type of herpes simplex, while in the more severe cases adenitis and fever are present. Draining lymph nodes are slightly enlarged and tender.



Fig. 276 Herpes Simplex.

dent with, or immediately following, the appearance of the eruption. The course of the disease is from five to ten days.

Herpes Genitalis In the male, the usual sites are the inner surface of the prepuce (herpes preputialis), glans, and shaft of the penis. In the female, the usual sites are the labia majora, the labia minora, vestibule, clitoris, and perineum. In this variety of herpes simplex, the lesions always rupture spontaneously and result in superficial abrasions. These abrasions may coalesce and form small circinate figures which are often secondarily infected. Healing occurs in from five to eight days. Recurrences are common.

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Treatment There is no specific treatment for herpes simplex. Wet dressings of aluminum acetate (1 per cent) sponging with hot water followed by spirits of camphor or the use of calamine lotion are effective. Equal parts of alcohol, glycerin, and tincture of benzoin compound have their advocates. Dusting powders like thymol iodide, zeroform, dermatol, powdered alum, and talcum are very helpful. Lesions of herpes simplex can be protected after rupture of vesicles by frequent applications of tincture of benzoin compound. Crusts are removed by applying boric acid ointment to which 5 per cent tincture of benzoin compound has been added.

Autovaccination has become a very successful treatment. This is accomplished by repeated intradermal inoculations of fluid contents of successive vesicles. Intradermal inoculations are made every other day.

Frequent local applications of spirits of camphor or a 2 per cent solution of salicylic acid or 5 per cent alcohol will often abort the eruptions of herpes simplex.

Recurring Herpes of Children

Some children are subjects of recurring outbreaks of herpes simplex on the cheek and chin.

The outbreak is sudden and free from prodromal subjective symptoms. It is characterized by a group of vesicles located on an erythematous base, commonly on or around the same area as the previous outbreak. The eruption usually subsides within a few days, leaving a slightly pigmented scar which may be mistaken for lupus vulgaris. Recurrences take place at intervals varying from

few weeks to several months. The etiology of recurring herpes is obscure.

Diagnosis The history of recurring herpes and the absence of characteristic jellylike nodules differentiates the scar of herpes from that of lupus vulgaris.

Treatment Treatment is purely symptomatic. Methenamine given at



Fig. 277. Recurring Herpes Facialis. Scarring is present and lesions recur on scarred areas.

the first sign of a recurrence and later over a long period appears to be of benefit at times. Initial doses of 2 gm (30 grains) every three hours for three to four days, should be given. The condition responds to x-ray irradiation and to autohemotherapy. Repeated vaccination with smallpox virus has been used to prevent recurrences of herpes simplex and recurrent aphthae, sometimes with success.

X-ray therapy It aborts herpes simplex lesions but it must be used within twenty-four hours of the onset of the lesions, in doses of $\frac{1}{4}$ erythema unit.

Herpes Menstrualis This is herpes progenitalis occurring during shortly before or after menstruation. The erosions resulting from this variety of herpes simplex are often very painful. The usual locations are the labia majora.

Recurrent Herpes Simplex Chronic recurrent herpes simplex usually occurs on the face, the lips, the buttocks, and the genitalia. Recurrence of facial herpes frequently follows exposure to sunshine and wind. The lesions in all varieties of herpes simplex heal without scar formation. Areas which were formerly the site of herpes simplex are usually the seat of brownish spots; these fade within a few days after the disappearance of the herpetic lesion.

Diagnosis Herpes simplex is readily identified by its characteristic lesions, the benign course of the malady and the tendency to rapid healing without discomfort or scarring. Pains and unilateral distribution of lesions which are absent in herpes *oster*. Macerated genital lesions of herpes simplex are not to be confused with chaneroid or chancre. *Chaneroid* is an ulcerative and auto-inoculable lesion attended with enlarged tender inguinal lymph nodes. *Chancre* is readily distinguished by the accompanying induration. However all erosive or ulcerative genital lesions are to be considered due to *Treponema pallidum* until proved otherwise.

Complications Herpetic lesions occasionally become hemorrhagic; this condition is then referred to as "black herpes." Gangrene may complicate this variety.

Generalized herpes of the face has been reported in France following infections as in diphtheria. Generalized herpes is ushered in with a fever of 104° F (40° C) accompanied by chills and pharyngitis. Herpetic vesicles over the

face occur within two or three days after the appearance of prodromal symptoms. The disease runs a course of four days and ends in recovery.

Prognosis Herpes simplex is benign and runs a course of one week or ten days. The lesions heal without scarring unless secondary infection occurs. Herpes simplex conveys no immunity; an attack predisposes to recurrences in the same location or in adjacent parts of the body. Some individuals suffer attacks regularly during certain seasons of the year or at intervals of weeks or months. A familial tendency has been observed in some patients, which is probably due to contagion rather than inherited sensitization. Recurring attacks of herpes simplex in the same situation may lead to telangiectases.

Prophylaxis Prevention of recurrence depends on discovery of the cause. Digestive disturbances, neuroses, foci of infection, etc., must be eliminated.

Scrupulous cleanliness is the best preventive measure against herpes genitalis. The genitalia should be washed daily to remove sebaceous secretions. This is followed by applications of a mild antiseptic lotion (2 per cent solution of boric acid). Thymol iodide and oleate of zinc are useful dusting powders. Ointments are contraindicated in the genital regions. Fractional roentgen treatment to areas which are the site of recurrent herpes is worthy of a trial. Large doses of thiamine hydrochloride, riboflavin, nicotinic acid, arsenic, and autolysin therapy are also prophylactic measures. Vaccination with smallpox vaccine performed every week for ten treatments has been successfully used in preventing recurrences. The use of several doses of snake venom (moccasin or cobra), given at weekly intervals, also delays recurrences.



Fig. 279 Herpes Zoster Ophthalmicus. (Courtesy of Dr. Carroll S. Wright.)

HERPES ZOSTER

SYNONYMS: *Zoster shingles, zona, ignis sacer*

Herpes zoster is an acute self limited inflammatory dermatosis characterized by the occurrence of groups of vesicles along the course of a peripheral sensory nerve. The vesicles are located on an erythematous and slightly raised base.

Varieties and Etiology *Idiopathic* herpes zoster is the most common form of the disease. It affects the cells of the posterior root ganglion and is probably

disease, vaccination and various blood dyscrasias. These pathological conditions are probably only predisposing factors.

Incidence The disease occurs at any age but it occurs less frequently before the age of ten and after seventy. It is rare in infancy.

Von Bokay suggested for the first time in 1930 the association of herpes zoster



Fig. 278: Herpes Zoster

due to an unidentified filtrable virus. *Symptomatic* herpes zoster is produced by action of various drugs as arsenical compounds, bismuth, lead, mercury, gold, morphine, carbon monoxide poisoning, etc. It may also arise in connection with involvement of the posterior ganglia in tabes, and from other disturbances of sensory ganglia produced by pressure of neoplasms, traumatic injury, and the toxins of chronic infections, such as syphilis, influenza, encephalitis, erysipelas, tuberculosis, leukemia, Hodgkin's

with chickenpox. Recent experiments show that the viruses of both diseases are identical.

Pathology The first lesions of herpes zoster occur always along the sensory nerve supply of the parts involved with the disease. The virus enters the sensory neuron, passes peripherally to the integument and centrally to the neuraxis. The formation of vesicles is due to direct action of the virus upon the integument. The posterior root ganglion is acutely inflamed with marked round

cell infiltration and hemorrhage. Degenerative changes in the posterior nerve root entering root fibers of the posterior columns of the cord and similar changes in the course of peripheral sensory nerves down the sites of eruption also occur. Vesicles are formed in the prickly-cell layer of the epidermis as the result of intercellular edema. The extent of the dermal lesions seems to parallel the intensity of the ganglionitis.

This antecedent symptom can become so severe as to be mistaken for angina pectoris, biliary colic, pleurisy or appendiceal colic. The neuralgia may precede eruptions by days, or even weeks, or may appear coincidentally with the skin lesions. A slight febrile reaction may occasionally occur before the appearance of eruptions.

Eruptions of zoster appear at first as red, grouped plaques which soon become



Fig. 230 Herpes Zoster

The protoplasm of the cells is opaque fibrinous, and vacuolated and the nuclei increased in number. This forms the balloon degeneration described by Unna.

Symptoms and Course. The disease is more common in adults. It is considered a bad omen in aged people because it is frequently a precursor of death.

The site of predilection is the lateral surface of the thorax. Herpes zoster does not always involve the spinal nerves; occasionally the Gasserian and geniculate ganglia are involved (orbital and supraorbital zoster). The disease is usually preceded by an antecedent neuralgia

the seat of vesicles. The vesicles are at first minute but they rapidly increase in size. The early lesions contain a clear serum, but after a few days the contents become purulent and the walls rupture and crusts develop. Vesicles occasionally become confluent to form lagoons of serum (herpes zoster bullorum). In herpes zoster hemorrhagicus, the serum contents of vesicles become hemorrhagic. The crusts are shed in a few weeks, leaving pigmented maculae. White spots occasionally appear at sites of former lesions.

While vesicles of herpes zoster never

cell infiltration and hemorrhage. Degenerative changes in the posterior nerve root entering root fibers of the posterior columns of the cord and similar changes in the course of peripheral sensory nerves down the sites of eruption also occur. Vesicles are formed in the prickle-cell layer of the epidermis as the result of intercellular edema. The extent of the dermal lesions seems to parallel the intensity of the ganglionitis.

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While vesicles of herpes zoster never

cross the midline a few aberrant lesions may cross beyond it along the course of some cutaneous nerve filament arising from the affected side. Occasionally bilateral occurrences of herpes zoster have been reported. Suppuration rarely occurs in herpetic vesicles but when it does occur the surrounding zone of inflammation becomes more intense. Le-

ulating chickenpox. This has been termed "generalized zoster."

Herpes zoster is accompanied by painful enlargement of the regional lymph nodes. Enlargement of the axillary lymph nodes accompanies zoster of the thorax and arms. The inguinal lymph nodes become involved in zoster of the lower abdominal wall or legs.



FIG 281. Left: Herpes Zoster. Right: Herpes Zoster and Varicella (gangrenous zoster).

sions of suppurating herpes zoster leave definite scars. *Zoster gangrenosus* or *necroticans* may occur in involvement of the first branch of the trigeminal nerve and elsewhere. In such instances, the cutaneous lesions are present for a long period of time and the subjective symptoms are severe. Lesions of necrotic herpes zoster leave definite depressed scars.

Occasionally vesicles occur on areas remote from sites of herpes zoster sim-

The preauricular lymph nodes are palpable in zoster involving the area of distribution of the trigeminal.

The sensory disturbances accompanying zoster vary in intensity. In children the accompanying neuralgia may be slight or absent. The intensity of pain appears to increase with age. The neuralgic pains preceding accompanying and following the appearance of eruptions may be so intolerable in aged individuals that the use of morphine be-

comes necessary. Pain may last many months after the eruption has disappeared. Paresthesia, hyperesthesia, and anesthesia may occur in the eruptive and post-eruptive stages of zoster and in the latter persist for years.

Diagnosis. The distribution and character of zoster lesions are such that no difficulty in diagnosis should arise.

Complications. Partial paralysis may follow an attack of zoster. The most common type of this paralysis is facial palsy following zoster of areas supplied by the trigemini. It is probable that the same toxic agents acting upon the sensory nerves involve the motor nerves. Paralysis of the extrinsic or intrinsic motor nerves of the eye occasionally accompanies or follows supraorbital zoster. Atrophy of muscles and loss of power occasionally follow zoster occurring on the arms.

Herpes zoster of the face may extend to the mucous membrane of the cheek, tongue, and throat.

The eyeball proper is occasionally involved in supraorbital zoster. Paralysis of accommodation, paralysis of the levator palpebrae superioris, and of other ocular muscles supplied by the oculomotor nerve (third cranial) may occur. The cornea may become involved and perforating corneal vesicles are often present. Iritis, choroiditis, and hemorrhagic retinitis are also complications. The vision may be permanently impaired or even lost. If the nasal branch of the ophthalmic nerve is affected ocular complications are likely to occur. The prognosis of herpes zoster is favorable for the eye and vision if there are no vesicles present on the nose.

Simultaneous attacks of herpes zoster are very rare, but they have occurred. More than one sensory nerve tract is usually involved in these rare cases.

Herpes zoster may appear on both sides of the body; however this is a rare occurrence.

An attack of herpes zoster usually confers an immunity against another attack.

Prognosis. The prognosis is always good in young and healthy adults.

The paralysis following attacks of zoster are not permanent.

Treatment. Constitutional treatment is directed to maintain the patient's vitality and resistance. Light nourishing food is given. *Acetphenetidine*, *sodium ashyolate*, *morphin* or *grains* may be administered to the point of tolerance for the relief of pain. *Methenamine* as a general anti-infective, has appeared to be beneficial in some cases. *Lumbar puncture* has been employed with advantage in very intractable cases of neuralgia.

Intramuscular injections of 1 cc. (10 minims) of posterior pituitary (obstetrical) double strength daily for three days may relieve the pain and shorten the course of attacks. Three injections are usually sufficient. *Pituitrin* should not be injected in pregnant women and those individuals suffering from cardiac disease or from hyperpnea.

The intramuscular injection of 10 cc (2½ drams) of the patient's own blood will often relieve the pain and probably shorten the course of the disease. It may be repeated in three or four days.

Intravenous injections of 10 cc. of 10 per cent solution of *sodium iodide* (*Ruggles*) given and repeated the second, fourth, and seventh day if necessary are highly recommended.

Large doses of *vitamin B complex* (100 mg. [1½ grains] daily for five doses)—especially *vitamin B₁* (thiamine chloride)—given hypodermically have given excellent results. *Cobra snake venom* and *Gnerygen* has been used with benefit.

Cutaneous areas are painted with flexible collodion or with liquid gutta serena to which is added 1 or 2 per cent of ichthyol. Calamine lotion containing 1 per cent ichthyol is recommended as a topical application when vesicles are large or have ruptured. Dusting powders of zinc oxide or zinc stearate dry the exudates from herpetic lesions.

Ointments and pastes have occasionally given relief. Lassara's paste with from 2 to 3 per cent of camphor has been employed. Lesions of zoster have been satisfactorily treated with a 10 per cent solution of picric acid followed by a dusting powder. Painful areas may be painted with a solution containing 1 per cent adrenalin and 3 per cent cocaine.

The application of a galvanic current of 1 to 3 ma. for a period ranging from five to ten minutes once or twice each day is beneficial. This is accomplished by moving the negative pole over the diseased area and placing the positive pole over the nerve trunk supplying the parts. Galvanotherapy is also useful in treating postzoster neuralgia.

Infected lesions are treated by ultraviolet light.

Pain is often relieved and the course of the disease may be shortened by unfiltered roentgen ray irradiation of involved areas in doses of 75 r ($\frac{1}{4}$ E.D.) filtered through 3 mm. of aluminum applied daily for three days to sensory roots of nerves at their emergence.

Ionization with sodium salicylate has been advocated. Trophic changes in muscles may be improved by massage and electrical treatment.

The following collumax is helpful in ophthalmic herpes zoster:

Acidi borici.	10
Sodii chloridi	10
Camphorae aquae	30
Aq. distill. q.s. ad.	100
Sig. To be used as eye wash every 10 or three hours.	

For the relief of pain in involvement of the cornea the following is advised:

Acidi borici	0.15
Sodii biborat.	0.15
Holocaine hydrochloride	0.15
Aq. distill.	100
Sig. One drop i. eye every few hours if necessary.	

Additional remedies for herpes zoster include quinine salts in large doses; this is especially indicated when constitutional symptoms are severe. Sodium nitrate (Chili salt-peter) 2 gm. (30 grains) may be given every four hours for relief of postherpetic neuralgia. Equal parts of a 35 per cent solution of tincture of aconite and chloroform applied over the affected area often affords prompt relief of postzoster pain.

Findler and Patzer obtained permanent and instant relief in four patients with acute painful zoster by infiltrating appropriate sympathetic ganglia with procaine hydrochloride (10 to 20 cc. of a 1 per cent solution). The regional sympathetic block (cervicodorsal and second and third thoracic sympathetic ganglia) was obtained by the anterior approach.

In thoracic types with intractable pain, sectioning the affected posterior spinal cord roots has been used, preceded by spinal anesthesia to determine the upper level of pain in the thoracic cord.

HIDRADENOMA

SYNONYMS *Syringocystadenoma* (Neumann) *syringoma* (Flocco) *hidradenoma eruptivum* (Jacquet and Darier) *lymphangioma tuberosum multiplex* (Kaposi)

This is a dermatoma characterized by small, benign epithelial newgrowths classified as sweat-gland adenomas.

Etiology *Syringocystadenoma* is ordinarily observed in young people between the ages of ten and twenty. The

discrete, oval, pale-pink or yellowish noticeably pinhead-sized to pea sized papules. The long axis of these slightly elevated papules often follows the lines of cleavage of the skin. They persist indefinitely enlarge very slowly but may even disappear spontaneously. Occasionally similar lesions are found on face, abdomen and arms. There are no subjective sensations.

Diagnosis They must be distinguished from *seborrhea*, which is yellow and from the lesions of *epithelioma nodosum cysticum*, which lack the softness, distribution, and color of hidradenoma.

Hidradenoma of the Lower Eyelids. This is a fairly common manifestation



Fig. 282 Hidradenoma. Of lower lids.

growths appear to have their origin in misplaced sweat-gland structures.

Pathology Histologically in the corium are found epithelial tracts of a more or less cylindrical form, having the diameter of a sweat duct, and being composed of elongated & polygonal epithelial cells. Cystlike formations are constantly made up of flattened epithelial cells, and are found 3 times to contain a colloidal substance.

Symptoms According to Darier there are two types: (1) a type described by him and Jacquet as *hidradenoma eruptivum*, and (2) *hidradenoma of the lower eyelids*.

Hidradenoma Eruptivum. In this rare type the lesions are found on the anterior surface of the chest and neck, and occur especially in women. They develop in the form of numerous, not very firm,



Fig. 283 Hidradenoma. Of lower lids. Note flesh-colored translucent oval papules on lower eyelids.

of sweat-gland adenoma. The growths are seen in the male, but more commonly in women of all ages. They generally develop after the age of twenty and show no tendency to spontaneous

Cutaneous areas are painted with *flexible collodion* or with *liquid gutta percha* to which is added 1 or 2 per cent of ichthyol. *Calamine lotion* containing 1 per cent ichthyol is recommended as a topical application when vesicles are large or have ruptured. *Dusting powders* of zinc oxide or zinc stearate dry the exudates from herpetic lesions.

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The application of a *galvanic current* of 1 to 3 ma. for a period ranging from five to ten minutes once or twice each day is beneficial. This is accomplished by moving the negative pole over the diseased area and placing the positive pole over the nerve trunk supplying the parts. *Galvanotherapy* is also useful in treating postzoster neuralgia.

Infected lesions are treated by *ultra violet light*.

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Ionization with *sodium salicylate* has been advocated. Trophic changes in muscles may be improved by *massage* and *electrical treatment*.

The following *collyrium* is helpful for ophthalmic herpes zoster:

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Sodii chloridi	10
Camphorae aquae	200
Aq. distil. q. s. ad	1200

Sig. To be used as eyewash every two or three hours.

For the relief of pain in involvement of the cornea, the following is advised:

Acid. borici	0.5
Sodium biborat.	0.12
Holocaine hydrochloride	0.05
Aq. distil.	150

Q. One drop in eye every few hours if necessary.

Additional remedies for herpes zoster include *quinine salts* in large doses; this is especially indicated when constitutional symptoms are severe. *sodium nitrate* (Chili saltpeter) 2 gm. (50 grains) may be given every four hours for relief of postherpetic neuralgia. Equal parts of a 35 per cent solution of tincture of *aconite* and *chloroform* applied over the affected area often affords prompt relief of postzoster pain.

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Fig. 283 Hidradenoma. Of lower lids. Note flesh-colored translucent oval papules on lower eyelids.

of sweat-gland adenoma. The growths are seen in the male, but more commonly in women of all ages. They generally develop after the age of twenty and show no tendency to spontaneous

disappearance. They are skin-colored only lighter or perhaps slightly yellowish so that they stand out clearly from the surrounding normal skin. Not infiltrated they are flattened but appear in most instances to be slightly elevated above the skin. This elevated appearance is lost on stretching the skin but the lesions themselves are not obliterated. There are no subjective symptoms, and on puncture one obtains only a drop or two of blood. They vary in size from a pinhead to about half a lentil seed. They consist of two or several lesions barely

visible or quite marked, on one or both lower lids. The lesions are soft and discrete and are multiple as a rule. The histologic appearance is the same as that of the first type.

They must be differentiated from *milia* which are firm and white and disappear on puncture and expression of their contents. The lesions of *milium* are definitely yellowish.

Treatment. The lesions in both varieties may be destroyed by *fulguration* or the *gallvanocautery*. X-ray radiation has been used with success.

HIDRADENITIS SUPPURATIVA

SYNONYMS: *Hidradenitis axill* *ls*, *abscess tubereux de l'aisselle* (*l'epaule*) *abscess of apocrine sweat glands* *apocrine acn* *apocrine furunculosis*, *hidradenitis destr* *as suppurativa* (Pollitzer)

Hidradenitis suppurativa is an inflammation of the sweat glands.

Varieties. Two varieties of hidradenitis suppurativa are recognized, one occurring in infants, the other in adults.

Incidence. Women are more often affected. The majority of patients range between the ages of twenty and thirty.

Etiology. The *Staphylococcus aureus* and the *streptococcus* are considered the causative agents. Infants suffering from rickets and other diseases of nutrition are frequently subjects of this disease.

Histopathology. The histopathology is characterized by an acute purulent inflammation with cystic distention of the sweat glands. The epithelial lining of the sweat gland is destroyed. Koch thought the sweat glands were more susceptible to infection because of the alkalinity of their secretion.

Symptoms. In infants, the lesions are more likely to occur on the back of the head, the back and buttocks. The lesions fluctuate and vary in size from 0.5 to 2.0 cm. in diameter. The super-

ficial lesions heal in a few days; however, when the infection involves the deeper tissues, large abscesses may result. In adults, hidradenitis is more often limited to the axilla. The primary lesion is a small, firm, tender nodule which at first is movable but later enlarges and fuses with the skin. The color of the lesions is bluish red. These nodules increase in number and form a cord-like elevated band. They finally fluctuate and open spontaneously. Extensive suppuration may result in ulceration which may be present for a long time. The ulcers show a granulating floor and a soft undermined ragged border. Nodules occasionally coalesce and form a flat purulent tumor with several openings. Persistent sinus tracts are usual. The subjective symptoms are pain and tenderness. This disease may involve the anus, nipples, groin, scrotum, and labia majora.

Diagnosis. Abscesses of hidradenitis suppurativa resemble *furunculosis* but are free of necrotic cores. The pain of

furunculosis is greater than in hidradenitis. The course of a boil is more rapid. The chronic cases may be mistaken for any of the infectious granulomata, mucocopic, serologic, and skin (Frei, tuberculum) tests may be needed for differentiation.

Complications. Gross deformity may follow extensive suppuration and ulceration of tissue. This is especially true in involvement of the groin and anal region.

Prognosis. The prognosis is favorable; however recurrences are frequent.

Prophylaxis. The avoidance of rubber dress shields, depilatories, and certain deodorants are prophylactic measures.

Treatment. The best treatment is a low-fat diet, the administration of thyroid extract, and roentgen ray therapy. Ointments are contraindicated. A lotion such as 1:5000 bichloride of mercury in 70 per cent alcohol, is useful for topical ap-

plications. Sulfanilamide internally and zinc peroxide externally are valuable remedies for treating burrowing ulcers. Ultraviolet-light therapy is valuable in infants suffering from this disease. Thorough incision in early cases, together with injections of whole milk is often valuable.

Frank C. Combes states that the local use of a bacterial filtrate made according to the method of Beredka has proved valuable in treating hidradenitis axillaris.

In some chronic cases, persistent abscess tracts should be widely opened and the wound thoroughly packed daily with gauze saturated in a 25 per cent aqueous suspension of microsulphathazole crystals. Penicillin 20,000 units intramuscularly every three hours for several days, should be given at the same time.

Total excision and plastic repair are occasionally necessary.

HYDROA VACCINIFORME (BAZIN)

SYNOPSIS. Summer eruption of Haeckelmann, hydron aestivale.

This affection is rare, congenital, and occasionally familial and its objective signs are produced by the action of sunlight.

Etiology: Although the exact mechanism for the production of the lesions is unknown, porphyria has been a frequent finding. The presence of photodynamic substances in the peripheral lymphatics appears certain. The eruption develops almost always after the patient has been exposed, for a variable period, to the sun's rays. The lesions develop the same evening or the following day and appear upon the cutaneous surfaces directly exposed to the sun. Although the eruption is generally observed from April to October it may in highly sensitive patients appear during the winter.

The disease first manifests itself during the first two years of life and tends to disappear in the adult state. Under normal conditions, photosensitive substances on which certain life processes depend are present in living tissue. Under abnormal or pathological conditions, an increase may occur in the amount, or perhaps in the development in vivo of a new photosensitive substance with resultant photodynamic effects from light previously innocuous. The rays in the solar spectrum responsible for this have been shown to be those at the short end of the visible and at the long end of the ultraviolet, light. The basic cause of these effects is said to be a photochemical reaction, the intricate nature of which, however, al-

though not understood as yet has been made the subject of considerable experimentation within recent years. Attempts to reproduce the lesions by artificial means have usually been unsuccessful. The work of Blum and his associates would indicate that the porphyrins are



Fig. 284: Hydroa Vacciniiforme

not the photosensitizing agents involved in the production of the lesions.

Symptoms The eruption may be preceded by general malaise, anorexia, nausea, and chilly sensations. More or less marked sensations of heat, burning, tension, and rarely itching may be present at the site of the eruption, which from an erythematous beginning rapidly becomes vesicobullous. The areas most often affected are the face, neck, ears, dorsa of hands, forearms, feet, and legs.

The typical eruptive element at the outset is a tense epidermic elevation or bleb, which is ordinarily surrounded by a red areola; the lesion may be rounded or oval, opaline or pearly, and may range in size from a pinhead to a pea. On pal-

pation it offers marked resistance. If punctured, serum exudes, but the epidermic elevation remains. In any case, it becomes yellow, then dry with a yellowish or sometimes black crust. The slightly darker central depression makes the lesion as a whole look umbilicated. When the adherent crust is removed at this stage, it uncovers a small, oozing ulceration. Left alone, these crusts drop off in from ten days to two weeks, leaving exposed a reddened, slightly depressed derm, which whitens slowly. If the lesions have been very large, the resultant scar is similar to those seen after variola. New lesions may develop on the sites of old ones, and in some cases telangiectases develop. The lesions vary from one or two to twenty or more.

During quiescent periods, scars on nose, cheeks, ears, and dorsa of hands



Fig. 285: Hydroa Vacciniiforme
Vesicular stage

are the only signs of the patient's light sensitivity. Hydroa aestivale is generally believed to be a mild form in which no scars occur.

Prognosis The skin tends to lose its photosensitivity with age. The general

health is unaffected, but in unrecognised instances marked scarring may occur.

Treatment This is largely prophylactic. Exposure to sunlight should be avoided, and the skin protected when exposure is unavoidable. The skin of the uncovered parts should be thoroughly smeared with a cream containing from

0.05 to 1.5 gm (10 to 30 grains) of quinine or salol to the ounce.

For the eruption itself a boric acid lotion—later cold cream or simple zinc oxide paste—containing 10 gm. (2½ drachms) of boric acid to the ounce is sufficient. The lesions heal rapidly irrespective of the topical applications used.

HYDROCYSTOMA

SYNONYM *Cyst of coil-gland duct.*

This is a rare noninflammatory disease of the face, characterized by discrete, persistent, deep-seated vesiclelike lesions.

Etiology Exposure to moist heat appears to be a factor as does excessive perspiration. The condition is almost always seen in middle aged women who have been working as laundresses or cooks. The lesions tend to improve during the winter. Site of predilection is the face (about the nose, eyelids, cheeks, and lower forehead). Histologically cystlike formations involving the sweat-gland ducts are found.

Symptoms The lesions are round or oval translucent, tense, and shiny elevations with a slightly bluish periphery. They range in size from a pinhead to a pea. They are firm and resistant to the point where more than slight effort is necessary to puncture them. On punc-

ture, a clear fluid appears, and the vesiclelike lesion collapses to form a sort of intradermic depression. When they dry spontaneously as is usual in two to three weeks, the end-result is a whitish spot suggesting a milium.

Diagnosis The location and character of the lesions differentiate them from sudanese vesicular eczema is inflammatory.

Treatment This consists—for those with a tendency to the development of the condition—in the avoidance of moist heat and the frequent application of a drying lotion, such as

Boracic acid	36
Boric acid	36
Zinc oxide	18.0
Prep. uracolumine	18
Witch hazel q. s.	100.0

Existing lesions may be punctured and the same lotion used.

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Vesicular stage.

are the only signs of the patient's light sensitivity. Hydroa aestivale is generally believed to be a mild form in which no scars occur.

Prognosis. The skin tends to lose its photosensitivity with age. The general

Parakeratosis is usually absent. The follicular orifices are usually dilated and contain a thick horny mass which may embody lanugo hairs.

The sweat glands and fatty tissues are only sparsely present. The sebaceous glands are atrophic and in some types they are entirely absent.

In the hypertrophic or *aunans* type, the epithelium may be either atrophic or hypertrophic and the stratum granulosum may be thickened or absent. The sweat and sebaceous glands are atrophic and the elastic fibers of the cutis are usually absent.

In ichthyosis hystrix, extensive horny plates are present which result from the increased production of horn. The stratum granulosum is entirely absent. The epithelium and cutis are atrophic. Elastic fibers are absent in the upper layers of the cutis and in the lower layer they exist only as fine filaments. The sweat, sebaceous glands, and the fatty tissue have almost entirely disappeared.

Symptoms. *Xeroderma (Xerosis)* This is the mildest form of ichthyosis. It is characterized by a dry and scaly skin. Xeroderma occurs frequently on extensor surfaces of the body on the scalp, and occasionally on the face. The palms and soles are dry and appear somewhat glazed. The normal folds of the skin are exaggerated. Xeroderma does not occur in the axillary and inguinal regions, nor does it affect the flexor surfaces of the knees and elbows.

Ichthyosis Simplex Ichthyosis simplex is a more advanced stage of xerosis and appears during the second year of life. It is characterized by a dry scaly and roughened skin which is dirty-gray in color. Its continuity is frequently broken by fine fissures. These fissures give it the appearance of reticulation. Secretions of the sebaceous and sweat glands are

either deficient or entirely absent. The extensor surfaces of the body are usually involved. Warty excrescences may be present on these surfaces in severe cases. The follicular orifices of the skin are filled with minute plugs which, when removed, reveal small curled-up hairs within them.



Fig. 287 Ichthyosis. (Courtesy of Dr. Jacques P. Gorzulewski.)

Ichthyosis Follicularis This is another form of ichthyosis in which hyperkeratosis is the most prominent feature. The skin has a rasplike feel. The scales are variable in size, thickness, and distribution. The scalp and eyebrows are usually dry and scaly while the hair is dull, brittle, and scanty. The skin of the face reveals dryness and scaliness. Slight ectropion may be present. The nails are often dystrophic, opaque, and ridged, and may appear elevated because of hyperkeratosis of the nailbed. The

ICHTHYOSIS

SYNONYMS: *Fish skin disease* alligator skin disease, xeroderma, xeroderma ichthyoides, hyperkeratosis universalis congenita, ichthyosis vera, ichthyosis simplex, keratoma maligna congenitale ichthyosis hystrix scuriala.

Ichthyosis is a congenital skin defect characterized by dryness, scalliness, and warty excrescences.

Varieties The several forms of ichthyosis include the following xeroderma ichthyosis simplex, ichthyosis follicu-

Etiology The disease is congenital. A hereditary tendency can be shown in some cases. The etiology is undoubtedly a prenatal change of the skin which causes hypoactivity of sebaceous and sudoriferous glands.



Fig. 286: Left Ichthyosis Simplex. Right Ichthyosis Hystrix.

laris, ichthyosis foetalis, ichthyosisform erythrodermia and ichthyosis hystrix.

Incidence Both sexes are equally involved. It occurs in every part of the world. It occurs more often in those countries where intermarriage is common. It appears in early life and becomes more accentuated during childhood and adolescence. It is less pronounced in summer and more marked in winter.

Pathology The histological report varies greatly and depends on the stage or type of the disease. In ichthyosis simplex, the cells of the mucous layer are smaller than normal. The rete cones are only sparingly present or entirely absent. The epithelium has the appearance of that of an infant or of an aged person. The stratum granulosum is everywhere absent and the horny layer shows varying degrees of hypertrophy.

Parakeratosis is usually absent. The follicular orifices are usually dilated and contain a thick horny mass which may embody lanugo hairs.

The sweat glands and fatty tissues are only sparsely present. The sebaceous glands are atrophic and in some types they are entirely absent.

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Fig. 287 *Ichthyosis*. (Courtesy of Dr. Jacques P. Gaezierr)

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tactile sense is more or less impaired. The mucous membranes are never affected in ichthyosis follicularis. Ichthyosis follicularis persists throughout life although the condition may improve in adolescence. It is always aggravated

by a shiny thick skin which cracks on the slightest body movement. Early death of these children is the rule, although they occasionally survive.

ICHTHYOSIS FOETALIS GRAVIOR. In this variety the infant is usually stillborn and, if prematurely born, it survives only for a few days. The skin is horny and thick, measuring from 2 to 3 mm. in thickness. Fissures are always present. These fissures may extend into the corium. The horny covering of the skin resembles armor plating. "Harlequin fetus" is the name often given to this type of ichthyosis foetalis.

Ichthyosiform Erythroderma. Ichthyosiform erythroderma is very similar to ichthyosis simplex but differs from it by the accompaniment of erythroderma and a generalized hyperkeratosis, more marked than in ichthyosis simplex. The flexor surfaces of the body are particularly involved and often show blackish horny vegetations. The clinical picture is one of an ichthyosis with erythema. The palms and soles are sometimes so extensively affected with this type of ichthyosis that the digits become contracted. The face is red and scaly. Ectropion is often present. The scalp is covered with sebaceous scales. Itching is usually an annoying symptom. The lesions are hyperidrotic rather than anhidrotic. Histologically according to Darier who considers the condition different from ichthyosis, all the epidermic layers are thickened.

Ichthyosis Hystrix. This is a rare form of ichthyosis in which the malady is limited to one or more parts of the body such as the arms, neck, and axillae. The condition is rarely universal. Warty or horny patches, varying in size and shape, characterize ichthyosis hystrix. The color may be yellow, yellowish-gray or green. Lesions assume an irreg-



Fig. 288. Congenital Ichthyosiform Erythroderma.

by cold weather and improves during the summer months.

Ichthyosis Foetalis. This is the most severe form of ichthyosis. It is fortunately very rare. Two forms of ichthyosis foetalis are recognized.

ICHTHYOSIS FOETALIS MITIOR. In this variety the infant is usually born at full term and is viable. The child presents

ular or a linear distribution and are the result of papillary and corneous hypertrophy. The affected surface is uneven, is more or less corrugated, and is occasionally covered with horny and spinous growths. These outstanding characteristics justify the appellation "hystrix." Ichthyosis hystrix linearis identifies a form of this malady in which the lesions appear in bands or streaks (see also Naevus, p. 526).

Diagnosis. The appearance of a well marked case of ichthyosis is so characteristic that difficulty in diagnosis should not occur. Only in the mildest cases should an error occur and then the history of the case, the lack of subjective symptoms, and the absence of inflammation should be sufficiently helpful in arriving at the correct diagnosis.

Complications. Chronic eczema may complicate ichthyosis. Psoriasis has also been reported as a complication. Ichthyotic patients are extremely sensitive to changes in temperature and are subject to frequent attacks of bronchitis.

Prognosis. Ichthyosis has no effect on general health or the normal span of life. The disease cannot be cured.

Prophylaxis. An ichthyotic patient should be told that it is possible to transmit the disease to his offspring and marriage should therefore be interdicted.

Treatment. Many authorities consider ichthyosis the result of hypothyroidism. There is no doubt that the long-continued administration of thyroid in fairly large doses causes improvement. It has also been noted that relapses occur as soon as the thyroid is stopped. Vitamin A in large doses (100,000 units daily) over a period of four to six weeks, is beneficial, especially in mild ichthyosis.

The use of parathyroid and calcium, pilocarpine or iodine has also been occasionally reported.

The diet should be rich in oily substances, such as cod-liver oil, olive oil, and should contain large amounts of vitamins A and D.

The patient will be more comfortable if a warm climate is chosen for a home.



Fig. 229 Ichthyosiform Erythroderma. More or less generalized erythema, with nonpruritic, persistent, chronic desquamation in patient who fifteen years later developed typical lesions of mycosis fungoides.

External treatment resolves itself in finding some suitable lubricant to keep the skin soft and to replace its natural oil. The patient should have a daily warm bath with a superfatted soap, afterward applying such lotions as cottonseed oil and solution of calcium hydroxide (equal parts) or olive oil (three parts) and glycerine (one part) or inunction of cocoa butter.

Hot alkali baths containing borax or sodium bicarbonate or colloidal baths of bran, starch or oatmeal are very soothing and pleasing to the patient. The

bath should be followed by an ointment such as

I	
Acid salicylic	1.25
Ung. quiphor	00 00
II	
Menthol	0 00
Plumb. celatry	0 10
Ung. a. sulphur	0 00
Glycerite of starch q. s.	30 00

George W. Andrews has the following statement to make regarding the treatment of ichthyosis. X ray and ultra violet light have no lasting effect upon ichthyosis.

"Temporary improvement frequently results from fractional roentgen therapy which is especially suitable when the condition has become eczematized."

IMPETIGO CONTAGIOSA

SYNONYMS: *Impetigo vulgaris*, *str. pyococci*, *Impetigo phlyctenular*, *Impetigo, staphyloidermi*, *streptoderma superficialis vesiculosa*.

Impetigo contagiosa is a contagious autoinoculable, inflammatory infection of the integument produced by streptococci, staphylococci or both. It is characterized by discrete thin walled vesicles or bullae. Vesicles undergo rapid pustulation and finally rupture. Rupturing pustules form slightly adherent crusts of amber or honey-colored tint.

Varieties. Several forms of impetigo are recognized, namely, impetigo bullosa, circinate impetigo, impetigo gyrata, furfuraceous impetigo, pemphigus neonatorum and Bockhart's impetigo.

Impetigo bullosa identifies bullous eruptions which rupture and leave raw crusted lesions. *Impetigo circinata* includes impetiginous lesions which are arranged in a circinate manner. *Impetigo gyrata* designates impetiginous lesions which spread by peripheral extension and heal from the center to form gyrate patterns. *Furfuraceous* or *dry impetigo* (circumscribed pityriasis simplex) identifies small irregularly rounded or polycyclic, persistent well-defined pink or skin-colored patches, with fine surface desquamation which commonly occur in winter. *Furfuraceous impetigo* is a superficial streptococcic infection of the face of children. Depigmentation is common when it occurs, as it often does, in the

Negro child. *Pemphigus neonatorum* is a form of bullous impetigo seen in the newborn. *Bockhart's impetigo* is a superficial staphylococcic perfolliculitis in which small pustules are seen at orifices of the pilosebaceous glands. The sites of predilection of Bockhart's impetigo are the scalp and extremities.

Etiology. The predisposing causes of impetigo contagiosa are age, want of cleanliness, pediculosis capitis, otorrhea and other foci of infection, and exposure to contagion. The disease may appear at any age. It is commonly seen during childhood and under the age of fifteen years. The healthy clean skin is rarely invaded by streptococci. Head lice induce scratching with eventual infection conveyed by the fingernails to the face and other exposed parts of the body. Herpes simplex about the mouth, seborrheic dermatitis behind the ears, and other conditions in which foci of infection are present may be complicated by impetigo. Contagion may be by direct contact with an infected individual or by indirect contact through contaminated towels, sponges, shaving brushes, and other toilet articles.

Histopathology. The histopathology of impetigo contagiosa consists of inflammation of the upper third of the funnel-



Fig. 290 Impetigo. (Courtesy of Dr. Carroll S. Wright.)

bath should be followed by an ointment such as

I	
Acid salicyl	1 25
Ung aquaphor	60 00
II	
Menthol	0 06
M. n. acetatis	0 12
Ung aquaphor	6 00
Glycerin of tannin a l	30 00

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shaped pilosebaceous follicle. Vesiculation lies between the prickle-cell layer of dermis and epidermis. Serous exudates contain leukocytes, streptococci, and staphylococci. The lymph spaces and blood vessels are dilated.

Symptoms The disease has no prodromal symptoms. It runs its course

adherent, giving a characteristic "stuck on" appearance.

The regions most commonly affected are the face, neck, and hands. Paronychia from contact of the fingers with facial lesions is common in children. The mucous membranes are rarely involved.



Fig. 291 Impetigo Contagiosa. *Left* Circular lesions. *Right* Note thick crusts.

without systemic disturbances. Itching is the only subjective symptom. The incubation period ranges from two to three days. The initial lesion consists of a transient erythematous macule. This is followed by the appearance of a flaccid vesicle measuring a few millimeters in diameter. The fluid contents of vesicles are at first clear, becoming purulent within from twelve to twenty-four hours. Vesicles enlarge, rupture, and dry to form characteristic semitranslucent yellow crusts free from any encircling inflammation. The periphery of a crust tends to curl while the center remains

Purulent folliculitis of scalp and beard results from staphylococcal infection of hair follicles.

The regional lymph nodes may be palpable, but not necessarily enlarged. A slight pyrexia may be present when lesions are abundant. The disease has a tendency to clear within one or five weeks. Healing occurs with eventual shedding of crusts which leave reddish patches. Patches disappear within a few days.

Impetiginization is a term used to indicate the characteristic signs of impetigo superimposed not as isolated

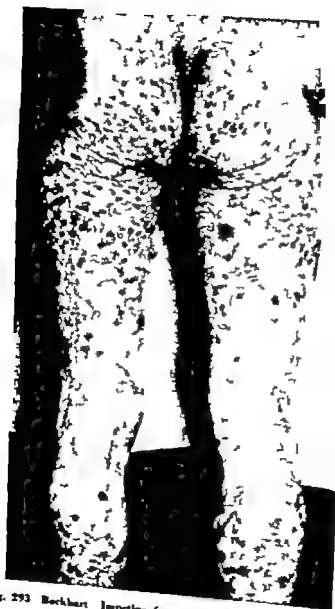


Fig. 293 Bockhart Impetigo (superficial pustular per folliculitis)

spots, but diffusely upon a preexisting dermatosis. The clinical picture is that of a crusted eczema which spreads peripherally although typical lesions may be present elsewhere upon the skin and which yields to the antiseptic type of therapy usually used in impetigo. Impetigo may become eczematized just as

patches of *tinea circinata* which possesses slight peripheral vesiculation.

Coalescing lesions of impetigo form patches resembling *pustular eczema* (eczematized impetigo). Such an eczema is accompanied by characteristic symptoms consisting of infiltration, intense itching, absence of rupturing vesicles, and a tend



Fig. 292. Impetigo Contagiosa. Bullous type; only one unruptured bleb; not the erosive nonulcerative remains of the others on same hand.

eczemas and dermatitides may become impetiginized.

Diagnosis. The lesions of impetigo contagiosa may simulate lesions of ring worm, pustular eczema, *ecthyma*, *varicella*, crusted lupus vulgaris, and tertiary syphiloderma. Circinate impetigo patches are more superficial and exudative than lesions of ringworm, and are covered with thick yellowish crusts. Furfuraceous impetigo does not resemble the scaly

ency to polymorphism. On the other hand, it should be noted that the lesions of impetigo are all alike.

Impetigo differs from *ecthyma* by the absence of inflammatory bases and areolae from lesions. Impetigo can be mistaken for early stages of *varicella*. The course and development of *varicella* is definitely pathognomonic.

Crusted lupus vulgaris and *tertiary syphiloderma* are attended by charac

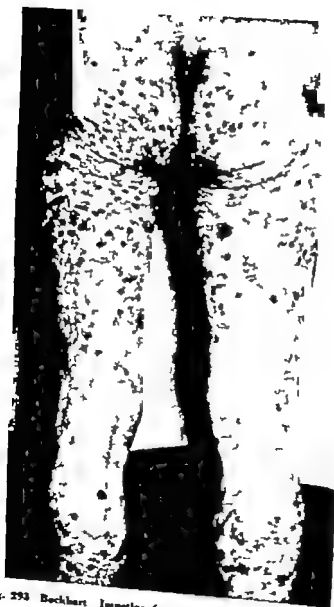


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Fig. 295 Pyoderma. A not uncommon form of chronic impetigo with slight tendency to deeper cutaneous involvement, pigmentary atrophy and even slight scarring.

teristic features of marked infiltration and ulceration below crusts.

Treatment Treatment consists of removing crusts as soon as they are formed without injury to the underlying regenerating epidermis, and the application of a mild antiseptic. Strong anti-

of sodium bicarbonate. Alibour water (eau d'alibour) may also be used. The formula for it is as follows:

Tr. saffron	0.5
Spts. camphor	5.0
Copper sulfate	2.5
Zinc sulfate	2.5
Aq. dest. q.s. ad.	1000.0
(alibourand)	

It may be diluted with water 1 : 1 four times, depending on tolerance of the skin.

Absorbent cotton wet with this solution should be rubbed into the lesion repeatedly until the crusts are off the eroded areas are then frequently dabbed with the solution or wet dressings of it are used.

The following paste is the best topical application to employ after removal of impetiginous crusts:

Hydargyri ammoniat	0.75
Pulv. zinc. oxid.	8.50
Pulv. amyl.	8.00
Acid. salicylic	0.50
Paraffini mollis q.s. ad.	30.00
Sig. Apply freely two or three times each day.	

Areas of impetigo in the scalp are aborted by applying on successive days a 4 per cent solution of silver nitrate in alcohol. Silver nitrate is, however, contraindicated for blonds because it stains the hair.

Hot compresses of boric acid are useful in treating impetigo of the eyelids and nares. Involved bearded areas are washed thoroughly in soap and hot water and followed by applications of 1:2000 aqueous solution of mercury bichloride. Shaving accessories are best sterilized by boiling.

Institutional patients with impetigo are isolated, their clothing sterilized and contaminated dressings burned. The daily use of ultraviolet light after removal of crusts is an adjuvant to treatment.



Fig. 294. Rockhart Impetigo
(aptylococcal impetigo) (Courtesy
of Dr. Ralph Bernstein)

septics tend to aggravate the condition by irritating the epidermis. Ointments are contraindicated when lesions are in the oozing stage. Lesions can be best dried by hourly applications of calamine lotion containing 0.13 gm. (2 grains) of precipitated sulfur to each 30 cc. (1 ounce) or by daubing with 10 per cent alcohol followed by a dusting powder like zoroform and aristol or by a single application of 5 per cent solution of silver nitrate. The drying of lesions is accomplished within a day of treatment. Crusts are removed easily by applying boric acid fomentations or by bathing involved areas with a 2 per cent solution

solution of *silver nitrate* with the following paste applied

Oil rose	10
Powd. zinc oxide	200

Baths of *permanganate of potash* 1:5000 are often effective.

An ointment of 5 per cent *amoniated mercury* or of 5 per cent *gentian violet* is combined with the treatment if fungi or yeasts are present. The acute

phase of intertrigo should subside before employing fungicides and antiparasitics.

In some instances, 5 per cent *sulfathiazole* in greaseless ointment base is specific within forty-eight hours.

In some bacterial types, the following is of great value

Fold thiazole suspension (20 per cent)	500
Witch hazel q. ad	1000
Rea. Apply three or four times daily	

INTERTRIGO

SYNONYMS: *Ch. fung. erythem. intertrigo*, *ecz. m. intertrigo*.

Intertrigo is an acute superficial dermatitis occurring on surfaces of the body which come in apposition.

Varieties Sabouraud identified two types of intertrigo (1) a microbial intertrigo which constitutes a streptococcal impetigo and (2) mycotic intertrigo which is a dermatomycosis.



Fig. 299: Inframammary Intertrigo.

Incidence It occurs more frequently in infants in persons who are obese and gouty, and in those who are sufferers from diabetes.

Etiology Etiological factors consist of hyperidrosis and maceration which furnish excellent media for bacterial and parasitic growth. Leukorrhea, urine discharge and uncleanness are also etiologic factors. Diabetic persons are particularly prone to attacks. The infecting organisms include the staphylococcus, streptococcus, yeast, and mycelial fungi.

Symptoms Intertrigo is usually accompanied by hyperidrosis and is characterized by redness, edema, exudation, crusting, and frequently fissuring at the bottom of the fold. An offensive odor

usually proceeds from the affected part. The secretion of intertrigo stains but does not stiffen the clothing of the patient, thus differentiating intertrigo from exudative dermatitis. Pustules and furuncles may also develop. The sites of predilection are behind the ears (retroauricular impetigo of Sabouraud), alae nasi from irritating nasal discharges, the gluteal and cruroscrotal folds, the inframammary region, the abdominal folds in obese individuals, the axillae and the folds of the neck. The oral commissures may be involved in an intertrigo known as "perleche." Subjective symptoms consist of sensations of heat and tenderness.

Treatment Prophylaxis consists of adequate cleaning of the body to remove perspiration, dirt and all irritating discharge. Opposing surfaces of the body are kept apart by gauze dusted with talcum or a tannic acid powder. The application of any powder should be preceded by vigorous rubbing in a solution of 1 per cent iodine crystals in 70 per cent alcohol. The following for *mulae* are also of value.

I	
Camphor	
Menthol	12
Thymol	0.5
Coal tar	5.0
Aluminum stearate	
Amylum a. q. s. ad	100.0
II	
Resorcinol	0.15
Lassar's paint	30.0
Sig. Apply on muslin for suborbital intertrigo.	

Suppuration is combatted by nonirritating antiseptics, such as gentian violet or 0.5 per cent lysol solution.

The affected parts are painted twice each week with a 5 per cent aqueous

cranial injection of vesicular fluid produces encephalitis in the mouse similar to that produced by the virus of herpes simplex. The virus scratched into the skin of a rabbit's ear produces no lesions, whereas vaccinia produces typical pustules. Patients who have recovered from Kaposi's varicelliform eruption can still be successfully vaccinated with cowpox (Barton and Brunsting). The disease is communicable.

Symptoms. Kaposi in 1883 described this rare complication of infantile eczema and, occasionally, of chronic adult neurodermatitis, seborrheic dermatitis, impetigo, and the puerperium. It appears suddenly with fever often high, and general malaise. The lesions, which tend to group or to coalesce, are discrete, lentil sized vesicles, often umbilicated. Many lesions may become pustular in severe forms of the disease. They appear on the eczematous surface especially but also on neighboring normal skin. They develop in successive crops over a period of from several days to a week. The affected area, usually the face, is red and swollen, and the draining lymph nodes become enlarged and painful. The mucous membranes may be involved. Epsels may be severe enough to cause death. In most cases the fever disappears by lymph

as the vesicles rupture and expose the corium or dry within ten to fourteen days, leaving pigmented spots and some varioliform pitted excoriations.

Diagnosis. This is usually easy in the fully developed case. The symptoms are rarely severe in varicella. In variola the eruption does not appear in crops. In dermatitis venenata and in iododerma the pustules are not umbilicated. Lynch states that in eczema vaccinatum the vesicles are apt to be fewer, larger and tenser and that more of them are pustular or umbilicated, the eruption and general illness endure longer. Experimental studies can be conclusive.

Prognosis. The disease is apt to be more severe in infants and young children. In infants, a fatal termination occurs in approximately 25 per cent of the cases.

Treatment. This is empirical. One should use local, soothing, mild antiseptic applications, as well as supportive measures when necessary. Wet compresses of a saturated solution of boric acid or a 1:4000 solution of potassium permanganate are satisfactory. Blood transfusions or injections of whole blood in the seriously ill have been suggested by Lynch. Sulfonamides have been helpful in some cases.

KELOID

SYNONYMS. *Hypertrophic scar, Keloid of Alibert, keloma, kelis, kelon, Kallenkrebs.*

Keloid is dermal neoplasm, usually following local injury.

Varieties. Two varieties of keloid have been described, the traumatic keloid and the spontaneous keloid.

Incidence: keloid is more common in males and most marked in those of the Negro race.

Pathology. Keloids consist of dense connective tissue bundles running paral-

lel with the surface of the skin. This dense fibrous tissue develops around the course of blood vessels. The clawlike prolongations of keloids are formed along vascular channels. Papillae and other epidermic appendages like the hair and sebaceous glands are absent in keloid formation. The rete malpighii runs as a thin continuous layer over areas involved in keloid. There is no anatomic

KAPOSI'S VARICELLIFORM ERUPTION

SYNONYMS: *Pustulosis vacciniformis (varielliformis) acuta (Juliusberg)*
vacciniform pyoderma.

Etiology This condition is for some the same etiologically and clinically as eczema vaccinatum (see p 371) for others, it is due to a filtrable virus probably related to the virus of herpes simplex. According to Lane Brunsting and Lynch there is in these patients, no history of a recent vaccination or of contact with someone who has been re-

cently vaccinated. Furthermore instead of cytoplasmic inclusion bodies (Guarnieri bodies) one finds, in tissue specimens, acidophilic intranuclear inclusion bodies in the patient's lesions and—if clear vesicular fluid is used—in the infected rabbit cornea indicating a virus similar to that of herpes simplex. O'Leary and Lynch state that intra-



Fig. 300 Kaposi's Varicelliform Eruption.

Surgery without irradiation does not give good results because recurrence is common.

Surgery combined with radiation gives the best results in radioresistant keloids. The radiation should be given at the first sign of recurrence rather than

and approximated by mattress sutures held in buttons to eliminate dead spaces.

Recurrence following combined irradiation and surgery is followed by the formation of young actively growing connective tissue cells which are radio-



Fig. 302 Keloid. Upper chest anteriorly. A common site. There was an associated nevus. Radium applications caused disappearance of the itching and almost complete flattening of the lesion.

before excision after excision. A suberythema dose of heavily filtered x-rays or Ir radiation by radium is given before excision. Excision is accomplished to obtain primary union. The wound is closed by a minimum of sutures. Small wounds are closed by skin clips. Very large wounds difficult to suture are restored by skin grafts.

Dorrance and Bransfield advise that the margins of keloids be undermined

sensitive to further irradiation by suberythema doses.

The technique of radiotherapy is influenced by the size, consistency, location, and age of the growth, and by the patient's age and the texture of the skin. Radium is applied only to hard lumps and cords which are not responsive to moderate roentgen ray therapy. Disfiguring keloids unresponsive to radium are best eradicated by the galvanocautery

pathological difference between the two varieties of keloid

Etiology The etiology of keloid is obscure although Hutchinson and others found a familial tendency. Keloids are rare in the aged and because of this, the hormones are thought to play a part in their development. Electrocoagulation by the high frequency current is often

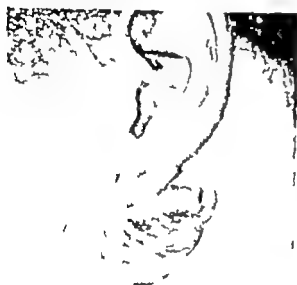


Fig. 301: A keloid. From ear stabbing

followed by keloid formation. It is for this reason that prophylactic x-ray therapy is advisable after this type of removal of nevi, verrucae or other neoplasms.

Keloids are known to follow mosquito bites, herpes zoster, acne vulgaris, vaccination, hypodermic injections, tattooing, perforation of the earlobe for ear rings, and syphilitic ulceration.

The spontaneous appearance of keloids is rare and probably due to pressure produced by corset stays and to the scratching induced by pruritus and by pin pricks or trauma so slight as to escape notice. The cutaneous surface over the sternum is a common site for this variety.

Symptoms Keloids are hard, raised, irregularly shaped and clearly defined pink nodules. They may appear singly or may be numerous. The surface of a keloid is smooth, shiny and firm. A characteristic feature of keloid formation is the clawlike prolongations arising from the central area of a keloid and extending into the surrounding normal skin. Clawlike prolongations from keloids however are not always present.

Keloids of mucocutaneous areas produce grave deformities. They impair joint movement if they occur over a joint. Keloids are frequently tender and may be accompanied by pain, burning and itching. The spread of keloids is by invasion of the normal skin surface for a distance varying from $\frac{1}{4}$ to 2 inches. The growth of keloids extends over months or years. They may remain stationary, spread very slowly and may occasionally undergo spontaneous resolution. Spontaneous resolution occurs commonly in children and following burns. Keloids rarely involve mucous membrane surfaces.

Diagnosis Keloids are differentiated from hypertrophic scars which are limited only to the original scar area. Keloids invade the skin beyond the area of scar formation.

Prognosis Keloids persist throughout life and are rather unresponsive to treatment despite the fact that few of them may undergo spontaneous resolution. Keloids are benign. Malignant changes in keloids are very rare.

Treatment The treatment of keloids is by irradiation with radium and heavily filtered x-rays. The earlier they are treated the better the results. Young growing erythematous keloids respond very readily to irradiation. Stationary keloids are old, less vascular and are not responsive to irradiation.

Ultraviolet therapy is of benefit when used for young keloids and when employed for improving the appearance of the skin after irradiation by radium and the x rays. Ultraviolet exposures are made to produce a second-degree reaction through blue cobalt glass.

Refrigeration by solid carbon dioxide, electrolysis, iontophoresis, and digestion by less active methods of treating this condition.

Palliative measures—consisting of massage with a 10 to 25 per cent ichthylol ointment, the daily application of 2 per cent menthol in cottonseed oil, or the use of a mercurial plaster—tend to reduce the pain and itching and aid in retarding growth of young keloids. Con-

tinued application of the following ointment is beneficial

Acid salicylic	8.55
Ichthyolla	4.00
Empl. plumbi	12.00
Empl. spathi	14.00
Glycer. pure q. ad	30.00

Rec. Apply on lint as continuous dressing

Injections of *thrombosane* and of *fibrolysin* are also helpful. One and one-quarter cc (20 minims) of a 10 per cent solution are injected around the growth at intervals of a few days. It can be given internally in daily doses of 0.2 gm. (3 grains)

Smyth suggests the injection of small quantities of *formalin* especially for treating keloids of the ear

KERATODERMA BLENNORRHAGICA

SYNONYMS *Gonorrheal keratoderma*, *gonorrheal keratosis*.

This is a rare disease occurring almost exclusively in the male and characterized by discrete or diffuse hornlike lesions on special areas of the skin, accompanied by arthritis. The arthritis is usually multiple and occurs during or after an attack of gonorrheal urethritis.

Etiology The causative factors are not known, and from a definite history of recent gonorrheal urethritis. The lesions have been interpreted as a manifestation of gonococcal septicemia, but the finding of the gonococcus in the affected joints or in the skin lesions has been infrequent. Both skin and joint lesions are most likely allergic expressions, in which the involved areas have been sensitized by a previous gonococcal infection and the usual secondary invaders. As rare as it is, it has become even more so since the introduction of penicillin in the treatment of gonorrhea and its arthritic complications.

Pathology Histologically the lesions,

when well marked, consist in acanthosis and marked keratosis. Early however there is rapid vesicopustulation, followed by crusting of a rupial character.

Symptoms The eruption is distributed symmetrically and commonly on the soles, palms, genitals, and groin less



Fig. 303 Keratoderma Blennorrhagica.



Fig. 303: keloid. In male following burn by fire.



Fig. 304: keloid. In female following application of pure phenol by patient to produce dermatitis factitia.



Fig. 207 Keratolytic Exfoliative Agents (Wende) (Courtesy of Dr. Carroll S. Wright.)

often on the scalp, dorsa of hands, feet, forearms, and legs. The lesions are often limited to the hands and feet—so-called localized forms. When first seen the patient usually presents isolated sometimes confluent hornlike brownish less often waxy crusts. When removed the undersurface is soft pink, and slightly

with the cutaneous lesions. Urethritis generally a minor feature.

Diagnosis. Keratoderma blennorrhagica is usually confused with *psoriasis* and *psoriasis arthropathica*. It is from the latter that this disease is sometimes most difficult to differentiate clinically or histologically. Keratotic cutaneous

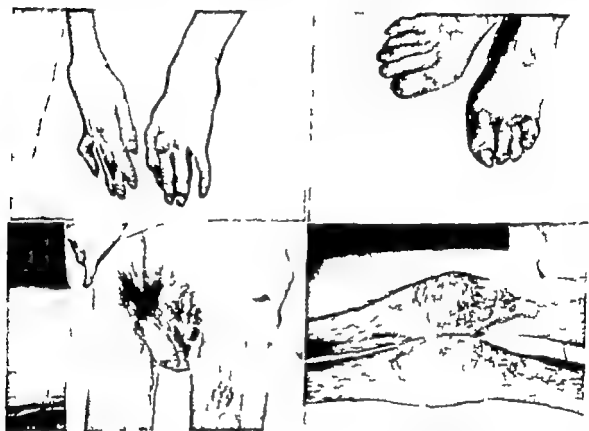


Fig. 306: Keratoderma Blennorrhagica. Note the swollen wrist joint and the hornlike lesions, particularly on skin over knees. There was marked cachexia and anemia. Penicillin (20,000 unit every three hours day and night up to 320,000 unit) improved skin condition but had no effect on the arthritis.

moist. Oral and corneal lesions have been observed. The nails are frequently involved. Pinhead sized vesicles and vesicopustules usually precede or accompany these hornlike lesions; chills and fever of a septic type are apt to precede each crop of new lesions. In the febrile cases, general debility, asthenia, and anemia quickly develop. Multiple very painful arthritis simultaneously develops

lesions, however associated with multiple painful arthritis should lead to an inquiry regarding recent urethritis; furthermore, chronic pustulokeratotic lesions are not characteristic of *psoriasis arthropathica*.

Prognosis. The disease is a more or less acute self limited process; the cutaneous lesions run a course varying from six weeks to eight months, with an

average duration of from four to six months. The arthritis is apt to persist long after the skin lesions have disappeared. Some of the reported cases have terminated fatally.

Treatment This consists in *supportive measures* to include a high caloric diet, high vitamin intake, iron, and liver extract. Blood transfusions may be necessary. Penicillin is of definite value as

a roborant and for its beneficial but incompletely curative effects on the skin lesions. A single course of 20,000 units every three hours, up to 320,000 units, is suggested. Penicillin has no effect on the arthritis. Massive doses of vitamin A (200,000 units daily) as well as *artificial hyperpyrexia* in conjunction with large doses of *sulfathiazole* have been used with success.

KERATOLYSIS EXFOLIATIVA (WENDE)

SYNONYMS *Desquamation cuticole ou alaire des mains (Corroyon)*

Etiology This condition is usually observed during the spring and summer and is not uncommon. It is most often seen in males, and especially in abortive forms.

According to MacKee and Lewis, it is probably a dermatophytid caused by the mosaic fungus transplanted from a focus of pathogenic fungi.

On histologic examination, Wende found a mild inflammatory process.

Symptoms Usually on both palms, sometimes on the soles, and on the dorsa of feet and wrists, there appear overnight one or several superficial, dry, white, circumscribed, pinhead-sized and larger sized, rounded spots. The central

parts of these spots break down and begin to desquamate. Apparently only the horny layer is involved. The lesions tend to extend, in a varying degree peripherally. New spots appear from day to day. There are no subjective symptoms, and grossly no inflammatory signs.

Prognosis Keratolysis exfoliativa (Wende) is a mild disorder which tends to disappear spontaneously. Recurrences at seasonal intervals and oftener are not unusual.

Treatment Therapy is generally unnecessary but a 3 per cent solution of *salicylic acid* in alcohol, applied several times a day appears helpful.

KERATOSIS

SYNONYMS *Keratoma senilis, keratoma seborrheica, verruca senilis.*

Keratosis is due to hyperplastic changes in the epidermis and mucocutaneous junctions.

Varieties Five forms of keratosis are recognized.

Keratosis Senilis. This identifies what is known as the skin of old age. It is characterized by graduated horny changes extending from smooth, freckle-like, pigmented macules to slight elevated, roughened areas. Fairly circum-

scribed, warty lesions arise resting upon chronically inflamed bases. The color of lesions changes from gray to brown and black. The size of involved areas varies from that of a pea to the palm of the hand. The consistency is hard. Lesions are firmly adherent and their forcible removal leads to bleeding. The exposed surface is moist. A patch of keratosis is not uniform in appearance because the newly keratinized periphery

average duration of from four to six months. The arthritis is apt to persist long after the skin lesions have disappeared. Some of the reported cases have terminated fatally.

Treatment. This consists in *supportive measures* to include a high caloric diet, high vitamin intake iron and liver extract. *Blood transfusions* may be necessary. *Penicillin* is of definite value as

a roborant and for its beneficial but incompletely curative effects on the skin lesions. A single course of 20,000 unit every three hours, up to 520,000 units, is suggested. Penicillin has no effect on the arthritis. Massive doses of vitamin A (200,000 units daily) as well as artificial hyperpyrexia in conjunction with large doses of sulfathiazole have been used with success.

KERATOLYSIS EXFOLIATIVA (WENDE)

SYNONYMS *Desquamation exfoliative en aires des mains (Cazayon)*

Etiology. This condition is usually observed during the spring and summer and is not uncommon. It is most often seen in males, and especially in abortive forms.

According to Macnee and Lowe, it is probably a dermatophytide caused by the mosaic fungus transplanted from a focus of pathogenic fungi.

On histologic examination, Wende found a mild inflammatory process.

Symptoms. Usually on both palms, sometimes on the soles, and on the dorsa of feet and wrists, there appear overnight one or several superficial dry white, circumscribed, pinhead-sized and larger sized, rounded spots. The central

part of these spots break down and begin to desquamate. Apparently only the horny layer is involved. The lesions tend to extend, in a varying degree peripherally. New spots appear from day to day. There are no subjective symptoms, and grossly no inflammatory signs.

Prognosis. Keratolysis exfoliativa (Wende) is a mild disorder which tends to disappear spontaneously. Recurrences at seasonal intervals and often are not unusual.

Treatment. Therapy is generally unnecessary but a 3 per cent solution of salicylic acid in alcohol applied several times a day appears helpful.

KERATOSIS

SYNONYMS *Keratosis senilis, keratosis seborrheica, verruca senilis.*

Keratosis are due to hyperplastic changes in the epidermis and mucocutaneous junctions.

Varieties. Five forms of keratosis are recognized.

Keratosis Senilis. This identifies what is known as the "skin of old age." It is characterized by graduated horny changes extending from smooth, freckle-like, pigmented macules to slight elevated, roughened areas. Fairly circum-

scribed, warty lesions arise resting upon chronically inflamed bases. The color of lesions changes from gray to brown and black. The size of involved areas varies from that of a pea to the palm of the hand. The consistency is hard. Lesions are firmly adherent and their forcible removal leads to bleeding. The exposed surface is moist. A patch of keratosis is not uniform in appearance because the newly keratinized periphery

is smooth and flat, while the older central portion is roughened and raised. Other senile changes of the skin consist of dryness, thinning wrinkling and hyperpigmentation. *Keratosis senilis* is not confined to old age because it may appear prematurely in early middle life. The areas of predilection are the face



Fig. 308: Keratosis Senilis.

neck, ears, dorsum of the hands and wrists, and areas exposed to climatic changes (sun, wind and cold). *Keratosis senilis* is more common in males because of their occupations which include sailors, farmers, mountaineers, etc. The condition predisposes to carcinoma.

Labial Keratosis This is closely allied to senile keratosis. It is characterized by the presence of scaly circumscribed keratotic areas on the vermillion border of the lower lip. Infiltration is absent in the early stages. It becomes markedly evident as the disease advances. The important features of labial keratosis are that it occurs in younger people and that its potential malignancy is far

greater than it is in senile keratosis.

Labial keratosis is differentiated from cheilitis by the sharper outline and the keratotic character of lesions, and from leukoplakia by the difference in color.

Tar and Oil Keratosis Continued contact with tars and oils lead to keratotic lesions which tend to undergo malignant changes. The condition occurs in workers who are engaged in occupations necessitating contact with these substances. The lesions are located on the arms, hands, and scrotum.

Arsenical Keratosis The ingestion of arsenic for long periods occasionally produces punctate hyperkeratosis of the palms and soles. These areas become thickened and present warty growths which may turn into epithelioma. The orifices of sudoriferous ducts are the sites of this condition (see also p. 283).

The body may also be the site of brownish pigmentation (arsenical melanoderma). The characteristic feature of pigmented areas is the fact that they are dappled by small light spots, hence the term "raindrop pigmentation." The pigment does not contain arsenic and is probably melanin. The mucous membrane of the mouth is rarely involved.

Keratosis Seborrhoeica (Senile Wart, Seborrhoeic Nevus, Seborrhoeic Wart) Seborrhoeic keratosis begins as small, oval brown thickening of the epidermis. While closely resembling senile keratosis, the condition differs in certain particulars. It may be observed in early life and more frequently on covered regions of the body especially in association with oily seborrhea. Lesions are always multiple and flat topped. The crusts and scales are admixed with sebum have a distinctly oily waxy friable character and a yellowish brown color. The site of predilection is the trunk. Senile keratosis has in contrast predilec-

tion for exposed surfaces. Lesions of keratosis seborrheica are numerous and may be arranged in a necklace fashion. The individual lesion is the size of a fingernail. They are popularly known as seborrheic moles. Itching is a prominent feature in some individuals.

Pathology The morbid anatomy is characterized by acanthoma, prolongation of the interpapillary bodies, and a rather regular cycle of keratinization. Small epithelial cysts are present, caused by the invagination of the epidermis. The acanthotic epithelium degenerates to form the greasy material which fills the interstices between the epithelial papillae and covers the affected area. The cutis reveals slight inflammation.

Prognosis Seborrheic keratosis rarely undergoes malignant change. If it occurs, it is usually of the basal-celled variety. Hazen states that 5 per cent of senile keratoses undergo malignant change. Senile keratosis of the face and extremities are more likely to result in prickle-celled epithelioma.

Treatment The frequent application of ointments such as ung. aquaphor or lanolin, and the avoidance of soap and hard water will benefit early lesions of keratosis senilis. Early lesions may also be removed with *trichloroacetic acid*.

Verrucous areas are removed by *fulguration* or *solid carbon dioxide*. Roentgen and radium therapy are also curative.

Labial keratosis responds favorably to the same treatment given for senile keratosis. The following ointment is beneficial.

Let. Plaster dust	50
Ung. aquaphor q. ad.	300
Sig. Apply to lips immediately after eating or drinking	

Tar and oil keratoses should be treated with *fulguration* or *roentgen-ray* or *radium therapy*.

Actinical keratoses are widespread and are treated by an ointment as

Acid salicylic	2.0
Aq. calcis.	2.0
Ung. aquaphor q. a. ad.	30.0
Sig. Apply twice each day	

Seborrheic keratoses are nearly always benign. Application of *trichloroacetic acid* is usually sufficient to effect a cure. *Solid carbon dioxide* or *superficial desiccation* is indicated for the elevated type of seborrheic keratosis.

Keratosis Follicularis

SYNONYMS *Dyskeratosis follicularis*, *acrodermatitis follicularis vegetans*, *Darier's disease*

Keratosis follicularis is a chronic cutaneous affection, characterized by crust-covered pinhead-size papules, which often are follicular and which join later to form vegetating patches.

Etiology In more than half the patients, the disease begins in childhood. It has a hereditary and familial tendency. It is seen in both male and female and often in association with endocrine disturbances.

Ford believes this disease results from two different types of genetic disturbances, (1) inherited as a sex-linked dominant in some, and (2) not sex-linked but an autosomal dominant in others. Pack and his co-workers believe it is a hereditary or acquired defect in vitamin A absorption or in conversion of provitamin A (carotene) to vitamin A.

Pathology: Histologically there is an increase in all layers of the epidermis, but the characteristic feature is the presence, often staining homogeneously of "round bodies" or dyskeratotic cells in the rete. Dyskeratosis may be absent in some of the papular lesions such as those on the dorsa of the hands.

Symptoms Darier's disease is usually symmetrical and ordinarily begins in the groins or on the scalp and temple. In any case, the usual localities are the articular folds (axilla and groin) the

observed over the dorsal surfaces of the proximal phalanges.

The early lesions are pinpoint, dry shiny papules scattered over an apparently healthy skin. They multiply in



Fig. 309: Keratosis Follicularis.

face, especially the temples, the scalp, especially the retroauricular areas, and the trunk, over the sternum anteriorly and over the nudthoracic area posteriorly. Horny follicular papules may be

crease slightly in size, and finally form a warty irregular patch. At this stage they have developed a strongly adherent grayish or blackish brown crust, which is somewhat soft and slightly oily to the

touch and which is found, when removed, to fit into a slight depression.

The lesions vary in color from dull brownish red to brown. In some the patches develop a vegetating character

scales; these give off a fetid odor and their surface may become fissured and eroded. The hands, feet, and tongue may become involved.

Subjective symptoms are variable



Fig. 310 Keratosis Follicularis.

with papillomatous elevations. Whether isolated or disseminated, they gradually become confluent, and the patches found are thick brownish-yellowish, flattened, and covered with horny or unctuous

chiefly pruritus, but the person's general health is good. The condition becomes worse in summer.

Diagnosis. The observer who has seen one well developed instance of

Symptoms Darier's disease is usually symmetrical and ordinarily begins in the groins or on the scalp and temple. In any case the usual localities are the articular folds (axilla and groin) the

observed over the dorsal surfaces of the proximal phalanges.

The early lesions are pinpoint, dry shiny papules, scattered over an apparently healthy skin. They multiply in-



Fig. 309 Keratosis Follicularis.

face, especially the temples, the scalp especially the retroauricular areas; and the trunk, over the sternum anteriorly and over the midthoracic area posteriorly. Horny follicular papules may be

crease slightly in size and finally form a wart-like irregular patch. At this stage they have developed a strongly adherent grayish or blackish brown crust, which is somewhat soft and slightly oily to the



Fig. 312 Keratosis Palmaris et Plantaris. Upper *Hand* at age of three. Condition present since birth. Lower *Left* Same patient at age of twenty-nine. Lower *Right* Same patient at age of twenty-nine.

Darier's disease is apt to recognize the next one without difficulty. However abortive, incomplete, or mild cases are not rare.

In *acanthosis nigricans* which may affect the same sites as keratosis follicularis, there is no scaling crusting or follicular plugging.

Treatment. Large doses of vitamin A by mouth (200 000 to 400 000 units daily) or by injection (100 000 units three times weekly) for a period of months are definitely beneficial to some patients. In those helped the drug must be continued otherwise relapse occurs. The lesions are also temporarily favor-

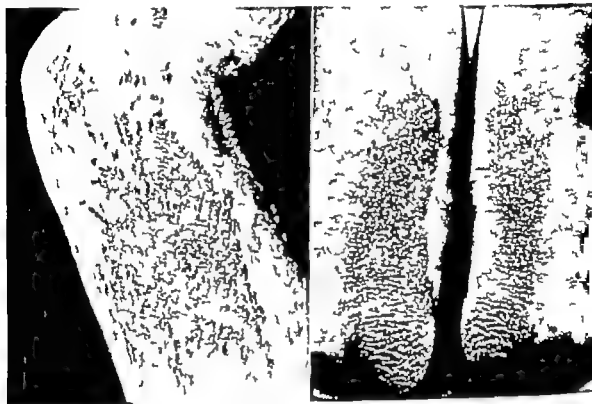


Fig. 311: Darier Disease (keratosis follicularis) Showing characteristic horny papules.

In *epidermodysplasia verruciformis* the resemblance clinically to papules of Darier's disease is close but dyskeratosis is absent histologically. Only cellular vacuolization occurs.

Acanthosis nigricans, *epidermodysplasia verruciformis*, *seborrhea* and *ichthyosis* appear at times to be closely related to Darier's disease.

Prognosis. Untreated the disease is progressive, and persists indefinitely. It often becomes generalized usually over a period of months or years. Regression is occasionally observed.

ably influenced by fractional x ray therapy. For other external therapy that recommended for psoriasis (p. 648) can often be used with benefit.

Keratosis (Palmar and Plantar)

This morphologic grouping is of practical value because several similar conditions develop on the palms and soles, and are at times clinically difficult to differentiate. This is particularly true of those characterized by from mild to severe thickening of the horny cell layer (hyperkeratosis). Darier classifies these kera-

13 *Keratoderma climactericum*. In 1934 Harthausen described a palmar and plantar circumscribed hyperkeratotic state observed in women, especially the obese and hypertensive during natural or induced menopause. The patches are thick, firm, and frequently fissured, and

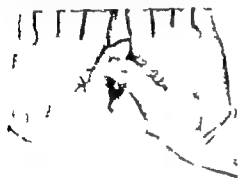


Fig. 814 *Keratoderma Palmare*. With similar condition on soles. (Courtesy of Dr. J. V. Klander.)

must be differentiated from hyperkeratotic ringworm. Itching develops if the rupture becomes eczematized. They are more often marked at pressure points. Prolonged estrogen and thyroid therapy as well as the roentgen rays locally have been used beneficially for this type of keratoderma.

Essential Keratoses 1 *Keratoderma punctata* (Hallopeau). This nevus state is often hereditary, persistent, and may appear at any age in either sex. It is characterized by hard, small, depressed or elevated, discrete, grouped or disseminated horny masses. They are the size of pinheads, greyish brown in color and leave pits when forcibly removed. This condition must be differentiated from the palmar and plantar keratoses found in arsenicism and Darier's disease, and from ordinary verruca.

2. *Keratoderma palmaris et plantaris* (Familial Keratoderma, Tylosis or

Ichthyosis Palmaris et Plantaris, Symmetrical Keratoderma of adults, Acrokeratoma, Meleda Disease)

Definition. A chronic, congenital, often hereditary sometimes acquired symmetrical inflammation of the palms and soles in which there is a red and infiltrative base, with a keratotic smooth or thickened horny epidermis of varying extent and degree.

Symptoms. The horny layer is smooth and soft or dry, firm, and thickened to a variable degree sometimes to the point



Fig. 815 *Keratoderma Climactericum*. In postmenopausal, forty-seven year old woman. The horny layer of the palms and soles was irregularly thickened. There was progressive improvement under synthetic estrogen therapy. (Courtesy of Dr. William Garbo.)

where it interferes with movement. Painful fissures of varying depths often complicate the hypertrophic changes in the horny cell layer. Portions of the skin or the entire skin surface of the palms and soles may be involved in the process. The

tooses as symptomatic and essential. The latter comprise two varieties, *keratoderma punctata* and *keratoderma palmaris et plantaris*.

Symptomatic Keratoses. These result from exogenous and endogenous toxic factors, localization of various dermatoses on the palms and soles, and repeated traumas, and may include

1 Arsenical keratosis (see p 283)

the dorsa of the hands and feet. The histologic appearance is characteristic and the presence of lesions elsewhere on the body aid in confirming the diagnosis.

5 Gonorrheal keratosis (see p 433)

6 Tinea keratosis in which minute vesicles may be found. In this condition, the scales show mycelial threads, and cultures are positive for tinea.

7 *Pityriasis rubra pilaris* (see p 619)



Fig. 313: Punctate Arsenical Keratosis.

2 Occupational keratosis, unilateral or symmetrical lesions due to repeated mechanical, physical, and chemical irritant contacts. These are actually callosities.

3 Keratotic chronic eczema due to a prolonged occupational irritant or allergic contact. Some vesicles and crusts are usually found.

4 Psoriasis of the palms and soles which may be unilateral or bilateral and be comprised of clearly defined circinate, scaly lesions which frequently spread to

8 Lichen planus. Typical lesions of lichen planus are found on the body and the microscopic picture is characteristic.

9 Psoriasisform syphilids of the second and third stages of the disease. Typical lesions of syphilis in the second stage are usually present on the body. The history is important and serological tests for syphilis are indicated.

10 Yaws and pinta

11 Ichthyosis

12 Congenital ichthyosiform erythroderma

even in its histological picture, in its epidemiology and in various other respects, it nevertheless shows no less numerous and important essential differences from that disease. One of these differences, fortunately for man, is the fact that, whereas tuberculosis affects mainly the internal organs, its ravages unseen and its victims tolerated by society the changes produced by leprosy are in the main manifest outwardly either as more or less conspicuous lesions of the skin, eyes, and upper respiratory tract, or as paralytic and destructive conditions secondary to involvement of the peripheral nerve trunks.

Ancient Records It is commonly stated that the most ancient records of Egypt, India, and China refer to leprosy the most commonly mentioned being the Ebers papyrus, of about 1550 B. C. Reference is usually made also to the Vedas of India, certain of which are supposed to be of the same antiquity and occasionally though more vaguely to Chinese writings. The role that the supposed references to leprosy in the Bible has played in our attitude toward the disease is notorious.

As to the supposed allusions to leprosy in the Bible, many writers have discussed the significance of the Hebrew word *sa-math* and have traced the history of the Greek term *lepra*. The former is undoubtedly generic, pertaining to a large number of chronic conditions affecting the skin, one of which may have been true leprosy. The latter signifies a scaling condition and is, therefore, more appropriate for psoriasis, and may have included leukoderma. In time, however *lepra arabum* came to signify our leprosy.

Nodular leprosy in Greece was later called *elephantiasis* whence the familiar *elephantiasis graecorum*, in the Byzantine

period *lepra* came to signify mutilating or neural leprosy.

Spread to Europe Regarding the spread of leprosy from ancient Greece, Egypt, and farther eastward to Europe, there are indications that it was present in Rome in the first century before Christ and in Gaul two centuries or so later. In the tenth and eleventh centuries, the disease was sufficiently prevalent in Scandinavia to be mentioned in laws promulgated in Norway in that period. By that time apparently the epidemic had come to affect all European territories.

Specially noteworthy is the more recent history of the disease in Norway where modern study of it began and modern control measures were first employed. During the first half of the nineteenth century there was a recrudescence, until in 1850 nearly 5000 cases were actually known. Under the measures introduced at that time, their numbers declined steadily and now there are only about 60 mostly very old. In Sweden, there are perhaps 10.

Spread to the New World The Americas, free from the disease when discovered did not remain so for long. The earlier explorers, or those who followed them, introduced it into Central and South America, but most important was the nonvoluntary immigration from Africa. The disease is now a major problem in Brazil, the Guianas, and Colombia, and it is increasing in Argentina, Venezuela, Ecuador, Paraguay and Uruguay have more or less numerous cases. It is of interest, however, that the native Indians who have not been in close contact with the new population are little affected.

In the United States, leprosy was introduced into the South from the West Indies as early as 1776. However the

LEPOTHRIX

SYNONYMS: *Trichomyces axillaris*, *Trichomyces palmellus*, *Trichomyces nodosa*.

Lepothrix is a saprophytic disease of the hair of the axilla and genital regions, and is characterized by firm yellow or black concretions on the hair or by soft sheaths surrounding them.

Varieties The several varieties differ only in color and are either yellow, red or black.

Incidence The disease occurs more often among those who sweat excessively and also more frequently in blonds. It occurs in all climates.

Etiology This disease is caused by the *Actinomyces tenuis*, also called *Nocardia tenuis*. The yellow nodes are produced by the *Actinomyces tenuis*; if it is associated with the *Micrococcus nigrescens*, the nodes are black; they are red when associated with the *Micrococcus castellani*.

Pathology On microscopic examination masses of mycelium in an amorphous substance (due to the pressure of trichomyces axillaris) are observed which penetrate the cortex of the hair. The mycelial threads are 0.7 microns in

diameter, nonbranching, regular and short and they form a resistant, horny glue. They never penetrate the hair follicle.

Symptoms Subjective symptoms are absent. The condition often escapes the notice of the patient. The hairs are brittle and often fracture. Lepothrix is usually present in the axillary region and less frequent in the genital region, the inner surface of the thighs, and the chest.

Diagnosis Diagnosis is established by microscopic examination of the hair.

Prophylaxis Prophylaxis consists in frequent bathing and the avoidance of filth.

Treatment Frequent bathing with soap and water and shaving of the hair are essential. The daily application of benzene followed by 2 per cent formalin or an alcoholic solution of 1/1000 bichloride of mercury is curative. Recurrences are likely and treatment should therefore be continued over a long period of time (see also Atrophy of the Hair p. 397).

LEPROSY

SYNONYMS: *Leprosy*, *Elephantiasis graecorum*, *Lepra arabum*, *Leontiasis*.

Introduction Leprosy, one of the most ancient of communicable human diseases, is today an active problem in many countries, though in others it is hardly more than legendary. In some leprosy regions it is probably no more prevalent now than it has been for centuries; in certain ones, it has been overcome almost to the point of extinction; in others, including parts of the New World, it is progressing. A conservative

estimate of the number of cases in the world is three millions.

It is no exaggeration to speak of leprosy as the most peculiar of human afflictions. For one thing, no other infectious disease is so sharply divided on clinical and other grounds into distinct forms or "types." Allied to tuberculosis in the general class of its causative agent, in its character of a chronic granulomatous process, and in certain forms

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Public Health Classification From the administrative, or public-health point of view leprosy cases are classified in a different way as "open" or "closed," according to whether or not they are bacteriologically positive. That feature depends upon the finding of bacilli in smear preparations made by standard methods from the skin or nasal mucus. Procedures like biopsy, nerve scraping, or lymph-node puncture are not involved, if bacilli found by such methods were taken into account, there would be few closed cases, indeed.

Causative Agent The causative agent of leprosy was first observed by Armauer Hansen in 1871. According to Loe, some thirty years previously Danielssen had recorded the presence, in preparations from leprosy lesions, of globular elements which he took to be cells and specific for leprosy but which later he described as yellowish-brown bodies of fatty nature. He stained the bacilli with aniline dyes and found them to be acid fast, the brown bodies he called globi. In the tissues they multiply for the most part within cells as if in a culture medium, both in the macrophages that undergo the modifications of the lepra cells, and in various fixed tissue cells. This micro-organism, *Mycobacterium leprae*, is beyond question the causative germ of the disease, despite the continued failure to fulfill Koch's postulates with it.

The leprosy bacillus presents the features common to the mycobacteria, notably the ability to retain dyes in the presence of acid and alcohol. It is readily stained by the Ziehl-Neelsen method and is gram-positive.

In staining smears with carbol-fuchsin, heating is unnecessary. The bacilli are well stained in fifteen minutes or less at ordinary room temperatures, and there

is an advantage in that they are often better colored and more plump than after heating. They are thus better demonstrated when small or fragmented.

Pathogenicity The pathogenicity of the leprosy bacillus is very low at least for adult man and for laboratory animals. In animal experiments, every conceivable route of inoculation has been used, with seldom any important indication of infection.

Among the more interesting observations are those of Nicolle, who found that upon repeated inoculation of a chimpanzee the sites of previous inoculations flared up as if sensitized. Others have had similar experience, those of Schöbl, Pineda, and Miyao being particularly interesting. The same allergic manifestations, however, have been seen in old sites of injection of the Mitsuda skin-test antigen (heat killed leproma) upon retesting or when lepra reaction occurred.

Under the circumstances, particular interest attaches to the attempts that have been made to inoculate human beings. According to Hansen, nearly thirty years before the discovery of the bacillus Danielssen serried in the belief that the disease is hereditary made several inoculations of himself and others, using leproma material with no untoward effects. Of the numerous deliberate human inoculations made since that time, one made by Arning in Hawaii in 1891, was followed by the development of the disease.

There are on record not a few circumstantial reports of accidental inoculations. The conclusion is that man can be infected by inoculation but that most people are resistant to the bacillus introduced that way as they are to exposure through contact with lepers; children, who in epidemic areas usually

disease is a very minor problem in this country confined mostly to parts of the Gulf Coast altogether there are perhaps 500 indigenous cases. It is endemic in Florida Louisiana Texas, and California.

World Distribution. As for the number of lepers in the world any statement contains too much of guesswork to be actually an estimate. Total figures now used by different writers vary from two to five millions. It is believed that China has a million and India no less. In view of a recent estimate of a possible half million for Nigeria alone and with indications of at least 150 000 in French possessions in Africa that continent may be credited with a third million. Allowing for possible exaggerations in those figures, to be balanced by the cases in other regions (hardly less than 100 000 in South America alone) three millions would seem to be a conservative total and it may well be far below the actual one.

Clinical Classification. The Leonard Wood Memorial Conference meeting in Manila in 1931 established a formal clinical classification which was improved and somewhat extended in 1938 by the Cairo Congress. The primary classification as it now stands and the definitions of "mixed" and "secondary neural" cases are as follows:

Neural (N) Type. All cases of the "benign" form of leprosy with disturbances of polyneuritic nature (i.e., alterations of peripheral sensation trophic disturbances, trophics and paralyses and their sequelae) or macules of nonlepromatous nature (i.e., lepraides, usually with localized sensory disturbances) or both. These cases give evidence of relative resistance to the infection are of relatively good prognosis as regards life although mutilation may take place and usually react positively to leprolin. Bacteriologically the skin lesions are typically but not invariably found negative by standard methods of examination, though the nasal mucosa may be found positive. Many of these lesions are histologically of tuberculoïd nature.

Lepromatous (L) Type. All cases of the malignant form of leprosy relatively nonresistant and of poor prognosis, usually negative to leprolin, exhibiting lepromatous lesions of the skin and of other organs, especially the nerve trunks. Bacteriological examination usually reveals abundant bacilli. Disturbances of polyneuritic nature may or may not be present they are usually absent in the earlier stages and present in the later stages of primarily lepromatous cases, and is often present in cases arising secondarily from the neural form.

Mixed Cases. Recognition should not be given to mixed leprosy as a type. However, cases of the lepromatous type usually exhibit, sooner or later varying degrees of polyneuritic involvement, and for practical work "mixed" or complete cases may be designated LN. The symbol "L" should be given precedence regardless of the original nature of the case or the relative severity of the two elements, because of the predominant importance of the lepromatous element. In grading the degree of advancement of these cases, the appropriate figure is placed after each symbol e.g. L2 N1 or L1 N3.

Secondary Neural Cases. Cases that have previously been of the lepromatous type with polyneuritic features (mixed cases) but in which the lepromatous lesions have resolved, leaving only the polyneuritic manifestations, are called secondary neural.

To facilitate expression of other features of cases in symbols, (1) the primary phase of mixed case may be indicated by LN or L¹N (2) a secondary neural case may be indicated as N and (3) the bacteriological status may be shown by adding to the main symbols either + or — or B+ or B—.

Subclassification is provided for in two ways (1) general, by degree of advancement as in the Memorial Classification each type being divided into three stages (symbols N1 N2, N3 and L1 L2 L3) and (2) special according to the nature of the lesions presented. The latter feature, a new one, pertains mainly to the neural type, which is subdivided into three varieties or forms (a) anesthetic (nonmacular polyneuritic, Na) (b) simple macular (with flat macules, N_a) and (c) tuberculoïd macular (N_t) further divided into minor and major

Pathology

Any discussion of the pathology of leprosy is complicated by the differences that exist between the two types of the disease. The discussion will be confined largely to the essential features of the lesions and their distribution, necessary to an appreciation of the clinical manifestations.

In the lepromatous type, there is universal dissemination of the bacillus throughout the body with important lesions in organs that are little or not at all affected in the neural one. In the leproma, the bacilli multiply as in no artificial culture medium yet devised, while the tissues remain relatively passive and deal with them essentially as foreign bodies, which is in keeping with the fact that the skin does not react positively to lepromin. In the neural type, on the contrary the tissue reaction is active, the growth and dissemination of the germ is strictly limited, and the deeper organs suffer little if any by direct invasion. The changes in the tissues that are affected, the skin and nerves, are, however variable in degree they may be, similar in nature to those seen in lesions of tuberculosis and sometimes in certain other chronic infections. These cases react positively to lepromin, papules being formed slowly that are of tuberculous architecture.

The classical division of the lesions of leprosy on "biological" grounds, set up by Jadassohn and expanded by Lewandowsky and generally accredited to him, is given by Janssens essentially as follows.

1. Xanthomas that are more or less well defined, very rich in bacillary masses and globi, resembling, like the milium follicles of tuberculosis and various mycoses, the form and structure of a granuloma. Examples: the lepromas of the skin, mucous membranes, nerves and deeper organs, in

which are found the typical cellular elements (*lepra cells*) and tissue structure of leprosy.

2. Inflammatory alterations, xanthomas and in no way characteristic, often consisting of loose perivascular masses of young cells and generally containing only scarce and isolated bacilli, the search for which is laborious. Examples: the initial lesions of the disease and noninfected nodules of the skin.

3. Lesions which histologically resemble more or less closely those of the lepra in Williams, the cutaneous nodule of Boeck and the hypodermic nodule of Darier and Reamy. Examples: tubercled lesions of the skin and nerve trunks.

The Essential Cell: The cell which appears in response to the presence of the bacillus in the tissues is the macrophage, of the reticulo-endothelial system. In the lesion it undergoes modification in either of two directions (1) in the leproma it becomes the well known and peculiar *lepra cell* (2) in the leproid it becomes the ordinary epithelioid cell and tends as usual to focal grouping. The former is quite definitely pathognomonic the latter is not.

The Leproma. The essential histological features of the lepromatous lesions are much the same wherever they are found. Essentially they consist of accumulations of macrophages that take up bacilli (though bacilli may also be found in fixed tissue cells and free) after which their cytoplasm undergoes a certain degree of increase in quantity and density to become "*lepra cells*."

The Leproid. Some of the older investigators saw no histological distinction between the leproma and the skin lesions of neural leprosy (called by Unna *neuroleprids*, an erroneous modification of Arning's term "*leproid*") Hansen and Looft wrote that "the maculae exhibit generally the same anatomical characters as do the nodular lepromata, viz., infiltrations with round cells, epithelioid and spindle cells. The difference between them is quantitative; in the macular

evidence most susceptibility to infection, are naturally not available for experimentation. It has been suggested that this situation may be due to lack of viability of the bacilli found in lesions, but it is much more likely that the explanation lies in the highly specialized biological requirements of this germ, and a natural resistance on the part of most living beings.

Transmission of Infection. Fundamental is the generally accepted principle that leprosy persons constitute the source of infection of other individuals, whether directly or indirectly and control measures are based on that principle. Another one that it is the bacteriologically "open" case which is dangerous. It may be agreed that as a rule direct exposure is required also that it must usually be prolonged.

With regard to the portal of entry the skin is generally regarded as the usual one, for example, the frequency of first lesions on the buttocks in children who are carried by leprosy mothers astride the hip is pointed to as an indication.

There is no necessity however to assume that a "primary" lesion must be produced at the point of entry. It commonly happens that patients claim first manifestations which indicate systemic invasion without any indication of where the entry was effected.

Susceptibility to Infection. A matter of the greatest importance is what happens once the infecting germ has entered the body. Granting the general predisposing influences of factors such as ignorance, poverty, crowding, poor nutrition, lack of sanitation and other primitive conditions, and of the existence of other disease, it is nevertheless a fact that under the worst of conditions many individuals, usually most of

them show a decisive resistance to the infection. On the other hand, well-to-do persons living under conditions beyond criticism may become infected. Children, relatively are highly susceptible, so that in leprosy institutions where children are born and kept, and also in many leprosy families, a large proportion may be infected. Because of this, they should be removed as soon as born. Among adults, the infection rate is low. Marital infection is infrequent.

Onset of Infection. From data published by Gomez, Avellana and Nicolas, Rogers and Muir worked out an average "incubation period" of 3.5 years for children born of lepers at the Cuban colony who developed the disease; had the period of observation been extended, to include cases that developed later that average would necessarily have been increased. For the run of cases found in the general population in endemic areas, such calculations are of little significance. In the end, however we arrive at the general and indefinite statement that as a rule several years elapse between infection and development of definite manifestations of the disease, though the period may be very short, as in the rare case in which the disease appears in infancy.

In view of all this, it seems more logical as in the case of tuberculosis, to look upon the interval between infection and onset as a *latent period* the termination of which may or may not depend upon some accidental precipitating factor than as an incubation period. That in cases, perhaps very many of them, there may be no progression from the latent stage to clinical manifestations (again as in tuberculosis) is suggested by individuals found to be bacteriologically positive in lymph node material but who remain healthy.

who were especially questioned by Rodrigues, one-third recalled paresthetic symptoms, numbness, tingling sensation, formication, darting pains, sensation of heat and cold cramps and rheumatoid pains in the extremities, another 10 per cent had noticed localized anesthesias, making a total of 45 per cent who recalled first symptoms that were referable to nerve disturbance. Not infrequently a patient claims, incredible though it seems, that the first sign of the disease was a trophic ulcer. It is said that pemphigoid bullae may be the initial symptom, but they are not common and usually they occur later.

On the other hand, in many neural type cases, the first manifestation is a small macule, well defined or hazy perhaps erythematous throughout, and at least in dark-skinned peoples, more or less hypochromic from the outset. Sensory disturbances when present, as they are sooner or later permit making the diagnosis quite definitely. The first really characteristic skin lesions of lepromatous cases are typically reddish or brownish and morphologically more indefinite than the leprids; if the onset is abrupt they may be multiple and disseminated. They are, of course, not anesthetic, but they are bacteriologically positive from the first.

Lepromatous Type

Lesions of Skin. Once the disease process is well established, cases of the lepromatous type are characterized primarily by skin lesions that, whether flat or otherwise, are more or less erythematous and diffusely outlined, often not delimitable, never showing central resolution. The actual color depends upon that of the individual, in the lighter skins, it is commonly darker than normal and slightly yellowish or brownish,

in brown peoples, the erythema may predominate, but in the darker skins as a whole the lepromata are usually somewhat lighter than normal, though typically much less hypopigmented than are the leprids. The surface is usually smooth often more or less shiny and with a velvety feel on palpation. These lesions do not exhibit the anesthesia of the leprids and usually are normal in this respect, though when infiltration is marked sensation there may cause some disturbance. A feature of the lepromas—as also of the leprids—is vascular instability manifested by marked color changes with change of temperature: reddish lesions often become bluish as they chill on exposure, and pale lesions may be made bright red by heat or friction. Bacilli are abundant and easily demonstrable. They may also be obtained at times from areas that show no evidence whatever of involvement, and the fact that when injections of methylene blue are given the dye may accumulate in unexpected places which is in keeping with other evidence that actual lesions are by no means always manifest.

In the macular lepromatous areas, the pathological condition is too slight to produce visible infiltration, but increased density is usually evident on stroking the area with the finger or picking up the skin. With progression of the disease, some or all of these lesions undergo general thickening, becoming morphological infiltrations and the surface becomes more smooth and shiny. The function of the sweat and sebaceous glands is interfered with sooner or later making the skin dry perhaps with slight parakeratosis, and the lanugo hairs may be lost entirely but such changes are less marked than in the lesions of neural leprosy. Finally whether on flat lesions

infiltration the number of bacilli are less." The question has been raised insistently if the active process in well established leprids of any class is ever *nontuberculous*.

In all of the leprids the intradermal nerve branches are particularly affected and the lesion often extends to the larger branches in the subcutis. Bacilli are more easily demonstrated in the nerves than elsewhere. Invasion of the nerves also occurs in lepromatous lesions of the skin but for some reason not adequately explained it does not result in loss of sensory function as in the leprids.

Distribution of Lesions *Lepromatous Leprosy* In this form of the disease the infection is systemic general, the bacillus being demonstrable at times in the blood. With respect to the occurrence of actual leprotic lesions and the effects produced by them however the organs are divisible into three groups: (a) those that are grossly affected and more or less seriously damaged; (b) those that are materially involved but not disturbed functionally; and (c) those that are not affected or if so not significantly.

In the first group fall primarily the skin, the peripheral nerve trunks, the mucous membranes of the nose, mouth and throat and the eyes. The testis is to be included here and also the superficial lymph nodes because of the degree to which they are affected though their function seems not to be disturbed. In the second group are the liver and spleen with the adrenals and spinal ganglia also to be mentioned. The third group includes the other visceral organs, the gastrointestinal tract, pancreas, kidneys, ovaries, etc.

Neural Leprosy In conspicuous contrast is the fact that only the peripheral nervous system, both the trunk and

cutaneous nerves, and the skin are much affected directly. Except for the nasal mucous membrane, which is sometimes invaded, such tissues are exempt, as are the eyes, except of course, for secondary changes consequent on paralysis of the lids. According to the variety of the case the trunk nerves alone may be involved or so far as definite manifestations are concerned only the skin and its peripheral nerves with changes in this matter as the disease progresses. The impression is prevalent that the more marked the localization in the skin, the less marked is that in the nerves.

Symptomatology

Onset The appearance of definite manifestations of the disease may be related to some unusual precipitating factor or circumstance. Among these are other diseases as malaria or physiological stress, as puberty or childbearing or perhaps even dietary disturbance or an event like chilling, exhaustion, or (some times heard from women) taking a bath at the wrong time of the month. Most often however the onset cannot be related to any such factor.

When the disease appears more or less abruptly as an acute "reaction," dissemination of the previously latent or indefinable condition, the symptoms are of the general class of those listed as prodromata but with definite manifestations involving the skin or nerves. Usually however the onset is insidious and variable. In many cases, regardless of type, there is given a history of areas of numbness either in the localized region supplied by a cutaneous nerve branch ("local") or of the extremities in the areas of distribution of peripheral nerve trunks ("polyneuropathy"). For example, of a group of 230 fairly intelligent patients at Cullin, all "open" cases,

Philippines. On the other hand in Japan, leprosy alopecia in advanced cases is very common and conspicuous, and is to be seen more infrequently in some other places. This is one of certain interesting regional differences of the disease another of which is seen in variations of frequency of blindness. The palms and soles are supposed not to be affected but histological examinations have shown them frequently to have

the effects of suppuration due to leprosy reaction. The lowermost one is typically largest, the higher ones smaller. Further evidencing progression upward, the iliac nodes may be affected by so to speak, overflow from below and in the extreme case the retroperitoneal nodes are involved as high as the kidneys. The portal nodes quite regularly show lepromatous accumulation, as may any that are in relation to the spleen, these being the



Fig. 317 Leprosy

some degree of lepromatous infiltration. Body areas that are more definitely immune to involvement include the axillary cubital, crural, and popliteal spaces.

Lesions of Other Organs. Organs Essentially Affected. Of the structures in the subcutis, the lymph nodes, mainly the inguinal and axillary groups, are early affected, sometimes the epitrochlears are also enlarged, but not the cervicals. The inguinal group especially may become massive, even to bulge in well nourished patients, but they always remain discrete in the absence of tuberculous adenitis or of—a rare condition—

two visceral organs most affected, but the mesenteric and bronchial do not. It may be added that the deposition of bacilli in the superficial nodes does not necessarily depend upon the existence of skin lesions below them; bacilli may be obtained from the inguinal nodes even when there is no involvement of the legs or as a matter of fact, no clinical sign of leprosy whatever.

The subcutaneous veins of the extremities may be considerably involved when there is marked infiltration of the skin of those parts. That condition presumably contributes to the circulatory defi-

or on definite infiltrations, more or less discrete *nodules* frequently appear be cause of curiously localized increase of the granulomatous process. Such nodulations may be so abundant and close-set as to be in effect broken up infiltrations. Very commonly this development is confined to the head and neck and the

sist, the effect often resembling a nodular condition and constituting the classical "*facies leonina*." The external ears become affected relatively early especially the lobes, and bacilli may be obtained from them even before there is any perceptible sign of local involvement. Sooner or later they become



Fig. 316 Leprosy (lepromatous type) With unusual involvement of ears.
(Courtesy of Dr. Howard Fox.)

extremities, especially the arms the skin of the trunk though perhaps affected throughout may be conspicuously free even from gross infiltrations. The subcutaneous nodules that sometimes develop on the extremities may be conspicuous or detectable only by palpation.

On the face where the distribution of the lesion process is especially asymmetrical, infiltrations are interrupted or broken by what natural lines per

thickened and may be grossly pendulous. A common early manifestation is thinning of the eyebrows, especially in their outer halves, and ultimately they are lost entirely. As the lids also become involved the lashes fall.

It has often been said primarily on the basis of European experience, that the hairy scalp is not affected in leprosy and practically speaking it holds true for many regions, including the

and throat are commonly invaded. Lesions of the larynx give the voice a characteristic hoarseness, and in cases may lead to chronic stenosis, aphonia and dyspnea. When these lesions undergo exacerbation in lepra reaction death may ensue from acute obstruction unless tracheotomy is performed.

The testis is always affected, regardless of age and the epididymis is often involved. The specific lesions of the testis are mostly in the interstitial tissue but bacilli and globi are often to be found in the cells of the seminiferous tubules and within their lumens, and consequently in the seminal fluid. In time spermatogenesis is abolished, and the testis undergoing a peculiar kind of fibrosis. Gynecomastia occasionally follows. The ovaries are not similarly disturbed.

Visceral Organs. One of the peculiarities of leprosy is the fact that the visceral organs suffer only slight anatomical and practically no functional disturbance, despite the fact that bacilli are more or less constantly being disseminated by the blood stream. The explanation of the fact is not evident unless it be that the bacilli find their temperature unfavorable. The liver and spleen commonly contain lepromatous accumulations, and they are often perceptible to the naked eye as small, uniformly distributed yellowish points; the related lymph nodes contain lepromatous deposits, as has been said. Usually the lesion foci in these organs are inactive, composed only of foamy cells. It is said that these organs may enlarge in lepra reaction. Small lepromatous accumulations are to be found in the adrenals, but so far as known they are without practical significance.

Lepra Reaction in Lepromatous Leprosy. The phenomena of lepra re-

action in this form of the disease are numerous and important. Long regarded it merely as an acute exacerbation of the disease process and ascribed largely to local and general effects of a hypothetical toxin accompanying the bacilli in their dissemination. It is now recognized as, so to speak, an immunological reac-



Fig. 319 Leprosy

dent. In some cases it seldom or never occurs; in others, it is frequent. More or less suddenly there is an acute outbreak which when severe enough to be febrile, is called "lepra fever" in that event other constitutional symptoms, as malaise and even rigors and prostration, may be present. In the lesser degrees of the condition, general symptoms may be absent or slight, the important features being the reddening and swelling of a few or many of the old lesions, or the appearance of new acute eruptions—usually with few bacilli at first—which

ciency that is so often evident in advanced cases

The subcutaneous nerves, though not entirely immune in this type are not affected to any material degree with the exception of the great auriculars. This fact together with the absence of localized anesthesia dependent upon injury

changes peripheral anesthesia of ascending or "acroteric" type, paralyzes and deforms, and trophic ulcerations, all quite as in the advanced stages of neural leprosy. Thus is reproduced the "mixed" or complete condition. If later the lepromatous element clears up as it may possibly do even without treatment,



Fig. 318: Leprosy

of the nerves in the skin constitutes one of the outstanding differences between the two types. The nerve trunks of the extremities, on the other hand are decidedly affected particularly at points of flexure over bone and under ligaments, easily detectable are smooth lenticular thickenings of the ulnar above the elbow often marked and of the external popliteal as it curves about the head of the fibula. At first this invasion causes no particular disturbance of function but in the course of time fibrosis is likely to take place, followed by polyneuritic

there is left the so-called secondary neural case

As part and parcel of the process, the eyes often become affected, with lesions of the cornea and iris but rarely of the retina itself and blindness not uncommonly results. Infiltration of and nodule formation in the nasal mucosa chiefly of the septum and lower turbinates, often leads to ulceration and secondarily to destruction of the cartilaginous portion (only) of the septum, with falling of the nose. The tongue, uvula, and other structures in the mouth

leprosy or of demonstrable nephritis, which is usually second, the patient becomes palpably depressed and unwell, the tone of the tissues and the circulation impaired. Leprotic ulcerations appear and become more and more numerous and refractory. Septic conditions often intervene, least frequently where the general care of the patients is best.

In patients who recover usually those who have been successfully treated, the lesions subside and if they have not been so marked or chronic as to lead to permanent thickening or loss of tone of the skin, and, of course, if polyneuritic changes have not occurred, the appearance of the individual may return quite to normal. As improvement progresses, the bacilli obtainable become constantly fewer until some, and then all, of the lesions give negative smears. Under proper regulations, the patient is still kept under treatment for at least a year before he is paroled, with repeated bacteriological examinations in which numerous specimens are taken each time from the nasal mucosa, the earlobes, and any areas elsewhere that seem at all suspicious. When a patient is released it is realized that bacilli probably still persist in lymph nodes and nerves (they have been demonstrated in persons dying ten years after all active signs have disappeared) and that relapse occurs in all too many cases; perhaps as many as 30 per cent.

Neural Type

The several varieties or subtypes are represented by cases with only polyneuritic manifestations (anesthetic neural, N) those with only simple macules (simple neural, Ns) and those with a variety of frankly tuberculoid leprosy of various degrees ("tuberculoid neural," Nt). The last group is sub-

divided into "minor" of the lesser grades, and major the more marked ones, often of reaction nature and quite frequently spectacular. There are cases that show combinations of different varieties of lesions.

The type may be subdivided into two main groups. The Cairo committee did



Fig. 321 Leprosy (maculoneurotic form). (Courtesy of Dr. A. C. LaBarre.)

that on the basis of presence or absence of lesions of the skin, recognizing anesthetic and macular groups. Perhaps it would be better to consider on the one hand, the anesthetic and simple macular groups—the neural ("maculoneurotic") type as the older writers understood it—and, on the other the clinically tuberculoid groups.

Anesthetic Subtype Cases of this subtype present "evidence of involvement of nerve trunks only (polyneuritic changes and sequelae) without skin lesions. The disturbances in their earliest stage may be difficult to detect; as Wayson has shown, a very early mani-

may subside completely or remain as permanent new lesions or both. These eruption lesions are often likened to erythema nodosum and the condition is called that—erythema nodosum leprosum. In severe reaction lepromas of the skin and even of the mucous membranes



Fig. 320 Leprosy

often ulcerate. The condition may be acute and brief or it may become sub-acute and more prolonged while sometimes in patients in bad condition it becomes chronic, protracted and completely disabling perhaps leading to death through "leprous cachexia." Other organs than the skin are often involved and acute neuritis, orchitis, and even arthritis and iritis may occur. These conditions are frequently agonizing and most difficult to deal with.

The condition seems to be of allergic or parallergic nature; probably identical with the *Hersheimer reaction* (*Hoffmann* and *Ramos Baer*) though recently it has been related to the *Schwartzmann phenomenon* (*Ermakova, Stem*).

Various things may precipitate a reaction as excessive (*chaulmoogra*) or unsuitable (*potassium iodide*) treatment, harmful exposures or excesses, and, many patients believe, certain foods. The condition sometimes follows small-pox vaccination and tuberculin testing. On the other hand it is often impossible to point to any apparent cause. As these reactions are often uncontrollable and cause permanent damage, most workers try to avoid them despite the fact that often conspicuous improvement occurs after they subside. *Hansen* and *Looft* pointed out that prognosis is best in patients who have had fewest eruptions," and many others have had the same experience. Recognition of lepra reaction is important in connection with treatment (care in employing *chaulmoogra* drugs, and special remedial measures) and for appreciation of the processes of the disease.

Termination of Lepromatous Leprosy. The duration of the disease in this type varies greatly in individuals, and also in groups under different circumstances: the averages may be from perhaps eight to fifteen years or more. Anywhere in its course progress may be interrupted and recovery take place, but that does not often happen except as a result of treatment, once the disease has become really advanced the prognosis is poor. Sooner or later after perhaps many years of relatively comfortable active life, the general condition of the patient usually deteriorates. Even in the absence of complicating tuberculosis, which stands first as the immediate cause of death in

burns, even perhaps rat bites) to the insensitive tissues.

Simple Macular Subtype The common case of this variety is at the outset, and ordinarily continues to be, decidedly different from the anesthetic one. Among early cases there are some in which the skin lesions, usually single, can be diagnosed positively because of the presence of the characteristic hypopigmentation and central or general hypoaesthesia, but often there are only less well developed ones that can only be classed as suspicious until further changes occur. Commonly there is slight coarsening of the texture of the skin, dryness, and a tendency to slight scalliness and loss of hair; marginal activity is manifested by slight erythema and usually a little infiltration, perceptible on palpation if not visually. These lesions, once believed by many to be of neurotrophic nature, are due to local action of the bacilli. Thickening of related cutaneous nerves is given much emphasis by workers in India, less by others, who do not see it as frequently.

In typical progressive cases, the slow centrifugal enlargement of the lesions continues, and usually others appear though, of course they may be multiple from the outset. This process may go on steadily for years, and with irregular progression of individual lesions in different directions and fusion of those that approach each other bizarre patterns may be produced, rarely most of the body surface may be covered. Affection of the mucous membranes is relatively slight, but bacilli may be obtained from the nose and ulceration may occur. It has been said that there may be anaesthesia here and that when facial paralysis is very pronounced the senses of smell and taste may be greatly diminished or lost.

The advancing edge of the skin macule may be very irregular perhaps streaming and with small spots in advance of it ("colonial" development) but more commonly it is fairly even and unbroken. Active leprods are typically well demarcated from the surrounding skin, most clearly so in dark-skinned peoples. Ab-



Fig. 223 Leprosy

sence of marginal erythema does not necessarily signify inactivity and cessation of progression. The process is rather suggestive of a slow grass fire; as the active zone pauses on the area behind it is left in a burned-over residual state. It is pale and anesthetic, and may show trophic changes. Subsequent recovery may be practically complete, particularly with regard to color but depending on the severity of the damage there may be more or less permanent changes; the epidermis may be thinned, shiny dry and thrown into minute folds (the

festation may be slight paralysis of the orbicularis oculi or of the lip. Commonly however the first disturbance is of the extremities, starting with anesthesia of the region of distribution of one nerve trunk as for example an ulnar or of several of them. The nerve trunk or trunks involved may be swollen



FIG. 322 Leprosy (maculoanesthetic form)

at first not however as much as may occur in lepromatous leprosy, but later fibrosis occurs and the nerves become smaller and abnormally hard. Typically the anesthesia slowly extends upward from the extremity to the elbow or knee or even higher. As this goes on hastened by attacks of painful neuritis if they occur atrophy and weakening of the muscles of the hand or foot develops, affecting in the former the thenar by pothenar and the interosseous muscles. The skin becomes atrophic and para-

keratosis is common. Occasionally there occur pemphigoid bullae that contain clear serous fluid perhaps to become purulent later but remaining free of acid fast organisms, they may clear up promptly lead to ulceration, or they may be followed by the appearance of macules.

Further trophic changes lead to thinning and contracture of the digits, especially the fingers (*clawhand* or *main en griffe*) and there may be uncontrollable flaccidity (*wrist-drop* *ankle-drop*). The late muscular changes have been likened to progressive muscular atrophy but with anesthesia. The bones undergo atrophy and radiographs show rarefaction, absorption and pathological fractures. By absorption or in part by extrusion of sequestra after necrosis the digits may shorten until they are completely lost. Such changes may be confined to one extremity or to a part of one (for example the two last fingers may be lost completely due to injury of the ulnar while the rest of the hand remains fairly normal) or all may be so affected that the patient becomes almost helpless. The nails become distorted and atrophic, but barring actual amputation residues of them will be found to persist even after the fingers have gone completely.

Various subjective symptoms are complained of as darting pains, evanescent disturbances of sensation and muscular weakness, tenderness of affected nerves may be marked. When to this picture is added facial paralysis following compression of the fifth and seventh nerves in their bony canals, with lagophthalmos, lacrimation and secondary changes of the eye (especially of the cornea) and perhaps mouth-drop and drooling the victim is most repellent and more or less helpless, and his difficulties are increased by frequent accidents (*trauma*,

even papulate) depending on the location of the granulomatous changes in the depth of the skin and their degree. Large lesions are often discontinuous, they may consist of linear zones, or be broken up or present gyrate or serpigin-



Fig. 324 Leprosy (neural type)
(Courtesy of Dr. Howard Fox.)

nous figures. As in the simple macular form, adjacent lesions do not overlap progression stopping on contact.

It is in these tuberculoid cases that growth of involvement of the local cutaneous nerves is most commonly seen. Further extension upward to the related peripheral trunks is not uncommon, and the usual secondary changes may result therefrom. On the whole, however there seems to be relatively little tendency to the production of polyneuritic manifestations in this form of the disease. Nerve abscesses occasionally develop in the grossly thickened and caseous tubercu-

loid nerves; they are of the "cold" type, produced by localized liquefaction. They may occur in practically any of the main cutaneous nerves and deeper trunks, and may be single or multiple even in a given nerve.

Lepros Reaction in Neural Leprosy
Lepros reaction is less conspicuous in the neural than the lepromatous type and has received much less attention, except recently with regard to tuberculoid reaction. Interpreting statements by Hansen and Looft, it appears that, though macular leprids usually appear stealthily they may also appear all at once with marked fever" and such patches



Fig. 325 Leprosy

may disappear in a few days; such eruptions are much less frequent and severe than in nodular leprosy. Though they used to be ascribed to a trophic origin the formation of bullae and the acute rheumatoid affections of the joints must

"crushed paper" effect) An interesting feature of these areas is the immunity to reinvasion that they have acquired. Where the progressing margins of two neighboring lesions meet, activity dies out because of unsuitability of the terrain beyond for its maintenance.

An interesting feature of this variety of the disease is palpable thickening of the superficial cutaneous nerves, perhaps with tenderness or tingling on pressure. Hansen and Looft noted that if carefully sought the finer peripheral branches may be felt as delicate thickened cords." With experience in searching for them they can often be found, though except for the great auricular notable disturbance of this kind is seen less often in other countries than in India. It sometimes happens that a thickened nerve is the only manifestation of the disease. Anesthesia in some part of its zone of distribution is the rule, but the area so affected may be so small as to be easily missed. Marked involvement of the peripheral trunks, on the other hand is common in cases of long standing with marked skin lesions, and that involvement leads to the usual polyneuritic disturbances.

Tuberculoid Subtype The group as a whole is characterized primarily by the presence of elevated leprids of highly varied morphology. The division into minor and major varieties is made in part upon the degree of infiltration but in the former there is a larger degree of morphological variation and much less evidence of a reaction factor than in the latter. The descriptions of these varieties adopted at Cairo are as follows.

Minor Tuberculoid. The lesions so designated are the less marked ones of the kind that has become generally recognized to be histologically tuberculoid and that is clinically recognizable as such with certainty. These lesions show definite elevation of characteristic appearance, though

there are considerable variations. They are usually marked by irregularity of the surface, due to the essentially focal nature and superficial location of the tuberculoid process. That condition may produce elevated bands or areas which may be continuous, or discontinuous even to the point of producing isolated papulations. Occasionally the process is relatively deep in the dermis, in which case the surface may be relatively smooth, and the appearance may therefore approach that of some of the major tuberculoid lesions, but the degree of the condition is less than in that form.

Major Tuberculoid The lesions so designated are the more striking grossly elevated ones in which recognition as tuberculoid has been largely confined in the past. They are major both in degree and nature of the pathological process. Typically the process invades the deeper layers of the skin to a marked degree and also the subcutaneous tissue and by further extension in the cutaneous nerves related to the muscles, it may produce gross involvement of them. Macules (leprids) of this variety are those most liable to be mistaken for lepromata, especially when they are (a) small but thick, morphologically nodular (b) in a reaction condition, reddish, turged and smooth or (c) bacteriologically positive. One feature that helps to differentiate them is their typically sharp demarcation and frequently asymmetrical distribution. Another distinguishing feature is the occasional tendency to the development of marked enlargement of the local cutaneous nerves, which condition sometimes extends to the main trunks of an affected extremity thus introducing a secondary polyneuritic element. A point of importance is the frequency with which these lesions start abruptly as a reaction condition, and the relative rapidity—and, sometimes, the completeness—with which they may subside.

In the great majority of cases, all those of the minor grade and also chronic major ones, the lesions spread centrifugally as do the simple macules. The active marginal zone remains elevated and conspicuous while centrally there is retrogression and healing often with more or less marked permanent changes. The outer edge of the elevated margin is abrupt, but inwardly it usually tapers off gradually. Thus are produced the annular leprids, of varied morphology (broad or narrow smooth of surface, or rough

tients often pass into a state of chronic invalidism, and with anemia and perhaps a low grade of nephritis; to quote Hansen and Looft, many die cachectic, without any definite cause of death to be found at autopsy.

Diagnosis

No clinical acumen is required to diagnose a typical, well-established case of leprosy of either type though it is surprising how long cases may sometimes

ing of bacilli, which ordinarily pertains only to the lepromatous type.

Bacteriological Diagnosis. The bacteriological diagnosis usually depends upon the examination of smears of material obtained from superficial lepromas, chiefly those of the skin since the nasal mucosa is seldom positive until the disease is well established. In neural-type cases, all lesions are typically negative though in the course of time nasal smears may perhaps become posi-



Fig. 227. Neural Leprosy.

go undetected even in leprosy regions. There is trouble with the earlier and less typical cases, but in leprosy regions the people themselves may recognize it very early; elsewhere even dermatologists sometimes have difficulty especially when undue dependence is placed upon the bacteriological examination. Even those most familiar with this disease come to a point beyond which they can only class a case as more or less suspicious, and there is no specific test to help them.

The cardinal signs of leprosy are (1) for neural cases, the presence of characteristic sensory changes in characteristic lesions, and, it is added by some enlargement of cutaneous nerves in relation to leprosy; and (2) the find-

ing of bacilli, which ordinarily pertains only to the lepromatous type. In reaction tubercloid cases, which are so easily mistaken for lepromata may be positive and even markedly so at the height of the disturbance. As a last resort, aspiration of an inguinal lymph node or exposure and scraping of an enlarged nerve trunk may be resorted to but those procedures are not practicable for routine work. The technique of obtaining material for the standard examination is important.

Technic. For making smears from the skin, two techniques have been advocated, the clip and scraped incision methods. In the former a fold of skin is pinched up and clipped off with scissors, and the raw surface of the specimen so obtained is rubbed on the slide. This

be included among the phenomena of this kind. Similarly with the changes induced in apparently quiescent macules when potassium iodide is administered as a test of activity in a new case or of cure in an old one in that event there may be only slight reddening of a narrow marginal zone, or the case may flare up to become major tuberculoid the new condition may subside quickly

become well established however permanent sequelae may be expected to remain after the infection has been overcome. Such effects are of various grades, mostly of polyneuritic nature.

In many cases in which the disease has been overcome there are sequelae so slight that there is no disability whatever. Many cases of course, are more conspicuous with actual mutila-



Fig. 326 Leprosy

or persist for long periods, or a nerve that had given no trouble may suddenly swell and become very painful and permanent deformity may follow. Exquisitely painful polyarthritides may occur. Thus is sometimes generalized and perhaps may leave permanent stiffness on subsiding.

Termination of Neural Leprosy
That the prognosis of neural leprosy is good so far as life is concerned has long been recognized. It is considered a self-healing condition. Once the disease has

tions, which they often try to explain as traumatic. Such patients are handicapped physically and also by the fact that they are doomed for the rest of a natural tenure of life still to be looked upon as lepers. Even the extreme wrecks may ordinarily anticipate many years of existence to which few object, and during which they can accomplish surprisingly much with what is left to them of their members.

By no means do all cases recover and complete their normal span of life. Pa-

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Fig. 227 Nerve Leprosy

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tive but the prominent skin lesions of reaction tuberculous cases, which are so easily mistaken for lepromata, may be positive and even markedly so at the height of the disturbance. As a last resort, aspiration of an inguinal lymph node or exposure and scraping of an enlarged nerve trunk may be resorted to, but those procedures are not practicable for routine work. The technique of obtaining material for the "standard" examination is important.

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tion By alternately making very light contacts with point and bread, discrimination between "sharp" and "dull" can be determined rapidly.

The temperature discrimination test is made by contacts with small tubes of moderately hot and cold water or with the special double-ended instrument designed for the purpose. This test is commonly neglected, especially under field conditions.

In connection with the examination of macular lesions, Rodrigues introduced a histamine test, the flare of which stops abruptly at the edge of a neural macule. This test is an interesting one, though not very widely used.

Other Neural Changes Thickening of cutaneous nerves in relation to leproids is stressed as a cardinal diagnostic sign by workers in India, as has been said. Not infrequently cases come up for diagnosis in which there are no skin lesions but sensory changes of an extremely referable either to a superficial nerve or to a peripheral nerve trunk. In the latter case, muscular atrophy and other trophic manifestations may be absent, and when present such changes may be of atypical location. On the other hand, neural affections other than leprotic, as, for example Bernhard's anterior femoral neuritis, may cause suspicious changes. Dry beriberi and syringomyelia are the most commonly mentioned conditions that have to be differentiated.

Dermatological Differentiation The list of dermatological conditions mentioned by various authors as possible of confusion with leprosy is a long one. It includes psoriasis, ringworm (*trich circinata*) *trich versicolor* leukoderma seborrheal dermatitis, scleroderma, erythema nodosum, and certain definite tubercula, yaws (which besides other lesions may produce gangosa and confusing plan-

tar ulcerations) tertiary syphilis, dermal leishmaniasis, and other conditions. It has even been argued that the causative agent of leprosy may share with other "viruses" in the production of the Beumer-Boeck sarroid affection. This list serves at least to emphasize how protean are the manifestations of leprosy.

Serology and Immunology The story of this phase of leprosy no less long and confused than that of its bacteriology must be passed over briefly. Though much work has been done with all of the serological reactions, using bacillus-rich lepromas antigens as well as many others, nothing has come that is of practical value. We have no specific serological test by which to diagnose early cases that cannot be recognized clinically or to determine latent infections in contacts, or—a matter of concern to the bacteriological investigator—by which to identify cultures supposed to be of the leprosy bacillus. Aside from the element of group reactions, difficulty lies in the fact that the organism of leprosy seems to have very little antigenic value, so much so that we cannot be sanguine that the situation will be materially improved when an unquestionable culture of the leprosy bacillus is made available.

Complement Fixation Reactions. Much work has been done in attempts to develop complement fixation reactions with variably treated acid-fast organisms obtained from lepromas or cultivated from leprosy and from other sources. Recently Gomes used the *Streptothrix leproides* of Deycke, and Lleras Acosta used his supposed leprosy bacillus culture. Both advanced claims, still to be confirmed, of high specificity and of sufficient sensitivity to indicate infection in persons with only suspicious lesions or none at all. The possibility that

method is painful, causes scarring and is not adapted to intensive and repeated examinations

The incision method now widely used is simple and causes minimal discomfort. Smears from as many places as desired can be made at a time and the examination can be repeated as frequently as desired. The skin is pinched up and a small incision made into the corium. Blood if drawn is wiped off and the little cut is scraped side and bottom. The small amount of fluid and tissue pulp so obtained is spread on a small area of the slide. Ten or twelve smears can be put on a single slide a material advantage both in staining and examining.

In making smears from the nasal cavity it is very faulty practice to take only surface material blindly on a cotton swab. The mucosa should be actually inspected using a nasal speculum and head mirror for any infiltration nodule or ulcer. Any suspicious area, or lacking that the septum is scraped with a small knife blade or other suitable instrument (as a Frear submucous elevator) to abrade the surface and obtain material from the submucosa. This examination is important in neural cases and in lepromatous ones becomes negative, because ulcers tend to persist.

In selecting points of skin lesions to be examined there is no difficulty with lepromata since they are diffusely involved but in dealing with the ordinary leprids attention must be concentrated on their marginal zones. It is perhaps well to include both earlobes in the examination of all cases, as they are sometimes found positive quite unexpectedly. It is, of course, important that instruments must be so cleansed that no bacilli can be carried over from one case to another or from one lesion to another in the same case.

Neurological Diagnosis. *Sensory Changes.* Loss of sensation in skin lesions, a characteristic of the centrifugally-spreading leprids, is very variable, more often only partial (paresthesia and hypoaesthesia) than complete (anesthesia). Loss of temperature discrimination ordinarily occurs first, but what the patient will notice is numbness. Development of these changes is rather slow and progressive and is more marked in the old healed centers of the lesions than at their margins, which in fact may be hyperaesthetic, tingling or even painful on tapping. Confusing the picture is the fact that in acute tuberculoid lesions sensation may for a time remain quite normal.

Perception of light touch is tested by applying a delicate object like a finely drawn-out bit of cotton wool or a delicate brush cut with scissors from a chicken wing feather or a very fine camel hair brush such as a Chinese "pen." Whatever is used contact should be made in only a limited spot to determine small affected areas or the precise limits of larger ones, and it should be very light to avoid confusion with deep touch perception. In dealing with stupid patients, and probably as routine with all, it is best to have them indicate the point of contact, when felt, by pointing with the finger. The patient's reliability is controlled by touching definitely normal areas occasionally and by calling for a response when no contact has been made.

Loss of pain sense may be so complete that a pin can be pushed deeply without protest, but usually it is only partial and superficial elicited by touching the surface very lightly with a sharp point. A needle or sharp pin thrust halfway through the rubber of a long pencil is a useful instrument for testing this condi-

tol suspensions of normal skin do not cause reactions. Results seem not to be influenced by the presence or absence of tuberculosis.

The reaction is looked upon quite generally as one of allergy but if that is so it is very different from all others in that class. It seems most logical to look upon this test primarily as one of ability of the tissue to react to the presence of the bodies of the bacilli—slowly to produce a papular lesion of the same essential character as that caused by the actual infection in the cases that react positively to the test. It is considered by some to be evidence of high resistance to infection, but that involves a large assumption and contrary evidence is accumulating. The only evident specific feature is the energy on the part of lepromatous cases, specific because they are still able to react to other organisms of the acid fast group. Interesting as the reaction is, it is obviously of no diagnostic value except in the limited sphere of classification of cases. In that connection, it may be of significance in lepromatous cases that are improving (becoming negative) or in the prognosis of clinically arrested cases (persistently negative ones being probably most liable to relapse).

Treatment

The treatment of leprosy has two aspects, general and special, neither of which can properly be neglected or subordinated to the other. General treatment, obviously consists of doing every thing possible to improve the general condition of the patient. Special treatment refers primarily to chaulmoogra medication practically everything else in the medical armamentarium having been tried and either abandoned or found to be of more limited value.

General Treatment The matter of general treatment need not be considered at length, being on the whole the sort of thing that is so essential in tuberculosis with the difference that exercise is called for more than quiescence. Any intercurrent infection or infestation that may be present or which may arise should, of course be eliminated if possible; such complicating conditions in leprosy present no peculiarity as regards their treatment, but they (notably active tuberculosis or nephritis) may preclude the employment of special antileprosy treatment. The diet should be adequate, sensible and balanced. Nothing special in the way of vitamin intake has been shown to be of any particular value in combating the disease itself, but there are various ideas current, some of them perhaps more than mere superstitions, about foods that are unsuitable. *Regularity of life* and avoidance of all excesses are important. For example, at the Kala Hospital, in Honolulu Wayson required his patients to observe the *asta* period religiously. At the Culebra colony which is so large and so much like a normal community that marriage cannot be prohibited, that event is deplored for young people who are doing well under treatment because of the frequency with which the disease progresses afterward.

Patients who can do so and those who cannot are beyond much hope of benefit from antileprosy treatment, should engage in some form of *regular activity* idleness of mind and body which encourages indulgence in introspection and self pity is harmful physically and as regards morale and discipline. Thus *occupational therapy* and *mental hygiene* have come to loom large.

Stein divides patients into five classes: (1) physically fit for most labor (2) of

the Witebsky-Klingenstein-Kuhn reaction for tuberculosis may be useful in leprosy is under investigation but while it is positive in large proportions of established cases, especially of the lepromatous type, it would seem to have little promise for actual diagnosis.

Nonspecific Reactions Many attempts have been made some of them extraordinary to arrive at useful nonspecific reactions. Probably all of those that depend upon globulin instability that have been brought out in connection with the diagnosis of syphilis and other wise have been experimented with. The most that can be said for the results is that they show the frequent existence of marked changes in the serum proteins, probably qualitative as well as quantitative. Considerable attention has been given to the erythrocyte sedimentation rate especially by Muir with decided limitations, it may be of value in the control of treatment. A peculiar test devised by Rubino is the precipitation of formalized sheep's erythrocytes by sera of lepers. It seemed so interesting that Marchoux and Caro took it up but general experience with it has not been encouraging.

Reactions for Syphilis Much interest has been taken in the relation of standard serological tests for syphilis to leprosy at first because that disease seemed to be the only important one other than syphilis in which the Wassermann reaction was often positive, afterward in connection with the question of the reliability of such tests in the diagnosis of syphilis in lepers. As technique became more refined, the proportions of "non-specific" reactions became smaller until various workers believed that methods like that of Kolmer and that the flocculation tests like that of Kahn, could be depended upon. Since then because

of inexplicably discordant results the matter has been thrown wide open again. In general lepromatous patients seem to have a tendency to give "nonspecific reactions."

Tuberculin Tests In the field of skin tests the tuberculin reaction in its several forms was tried out very promptly after it was devised. Here interestingly enough there is no difficulty it has proved to be as specific for tuberculosis in lepers as in other people. The only possible indication of a cross relationship with leprosy is the fact that a small proportion of any group of patients so tested is liable to come down with (parallel) lepra reaction.

The Lepromin Reaction The so-called lepromin" or Mitsuda reaction is by far the most interesting test in sight. That investigator began about 1918 to use intracutaneous injections of saline suspensions of killed bacilli rich lepromas, and in 1923 he described a reaction that was unlike that caused by tuberculin or any similar antigen. It is not read after one or two days, but after two or three weeks, and the reaction lesion is a solid papule that may persist for months. Peculiarly it is typically negative in the lepromatous cases, in which the bacilli are so abundant but positive in neural cases—most strongly in the tuberculoid varieties—and also in most supposedly normal persons, at least after infancy.

Lepromin filtrates give negative results, and consequently it is the general belief that the formed organism is necessary to produce the reaction though question has been raised on that point. Lepromins prepared from rat leprosy lesions cause positive reactions, in this case, the lepromatous cases also react, and the same is true when suspensions of saprophytic acid fasts are used. Con

Iodized, were adopted for routine work. The iodine, which was used in Hawaii with the idea that it might be of therapeutic value, was decreased from 4 per cent to 0.5 per cent, its only virtue being in some way to reduce the irritant quality of the esters. Creosote (usually 4 per cent) is sometimes used for the same effect, and benzocaine (1 per cent) is similarly employed at the Carville leprosarium. In some places, particularly bland ethyl esters are used alone without additions.

Equally effective as *H. wrightiana*, of southern India, is *H. anthelmintica*, of Siam and Indo-China. Both species are abundant and the oils are obtainable fresh and at low cost. Pure oil of low acidity being thus available or easily prepared, that material is used by some instead of the esters, but in many places the latter continue in preference. Use of the present form of the sodium salt, called *alepoi* is at present limited. Given intramuscularly (3 per cent solution) it is painful, and intravenously (1 per cent) it often causes sclerosis and occlusion of the veins. In these dilute solutions, the amount of actual drug given per dose is so small that, even if this form of it were specially active, the balance could not be in its favor. The possible superiority of a chaulmoogra-cholesterol complex prepared by Baranger is under investigation.

METHODS OF ADMINISTRATION Of the methods of administration of either esters or oil, the intramuscular route is the most generally used. The subcutaneous one has never been widely favored, though it is still recognized. The intravenous route has been employed but little, and is not recommended because of serious disadvantages.

An important advance in treatment was made in the Philippines after inves-

tigation of the so-called "plancha" method which inmates at Calson had been using among themselves clandestinely consisting of injection of chaulmoogra esters intradermally into the lesions. As developed by Lara, this method was soon taken up in India and subsequently in many other places. The Cairo committee attested the fact that it is of special value in treating the lepromatous form of the disease, and desirable in the tuberculous forms except in its acute phases.

The method consists of infiltrating the area or areas to be treated by multiple small injections placed sufficiently close together for coalescence by diffusion. The needle—a rather small, short one is preferred—is inserted into the carinae and about 0.1 cc. is injected to produce a thick wheal; if the drug is placed fast beneath the epidermis adhesion will occur. As many as 100 punctures may be made at sitting, but it is advised that not more than 8 cc. be administered in this way at a time. Lows recommends usually not more than 2 to 3 cc. In practice, part of the desired dose is usually given intramuscularly though it is to be understood that the intradermal method has more than local effect since much of the drug put into the skin is mobilized and transferred for service elsewhere.

The injection of given area is not repeated until the inflammatory reaction that it causes has subsided completely and preferably not more often than once a month. An occasional case will be found to be unsuitable because of excessive inflammatory reactions. Whole oil of good quality may be nonirritating, but it is less suitable than the esters because it is less readily absorbed and tends to make the tissue boggy. Iodized esters are usually used in institutions, but the dark and often persistent discoloration caused by the metal anodes the crescent product preferable for use elsewhere. Even in nonanesthetic (hypersensitive) lesions, the method is not so painful as might be expected.

The special virtue of this method is not solely dependent upon the local irritation and inflammatory reaction that it causes. A part of the drug is taken up by the lepra cells present, and the effect on the bacilli in them is, therefore,

moderate working capacity (3) fit only for light work (4) capable of taking care of their own quarters and food and (5) no work possible or desirable. Specially insistent with regard to physical condition Muir goes so far as to say that "no real improvement can be made through any yet known drug apart from raising and maintaining the general health of the patient" and he has advocated postponement of special treatment until the patient can be brought to a first-class physical condition. Ryrie insists that exercise should be regular and moderate and that violent exercise and physical and mental strain may be harmful a warning that is supported by the history of the athletes among the patients at Culion.

Special Treatment. Chaulmoogra oil including of course its derivatives, is the one drug that has come into and remained in general use, from a very ancient beginning it is said to have been used over 500 years ago in China and long before that in India. The opinion that its use is of value does not receive support in all quarters, there being a seemingly inexplicable divergence of opinion in this matter. At Kalih: from where McDonald and Dean drew attention to the value of the ethyl esters about 1920 Wayson came to the conclusion that general treatment alone did all that could be done and a colleague of his once suggested that if a placebo must be given to lepers, something simpler and more bland should be used. Yet at the U. S. Federal Leprosarium where the patients have much to say about what treatment they will undergo they continue freely to take such drugs as if they were beneficial. Experience in most leprosy regions has been such that the modern chaulmoogra preparations are being used more and

more though as stated by the committee on treatment of the Cairo congress no form of treatment as yet available is wholly satisfactory.

Chaulmoogra Oil and Derivatives Used. For many years chaulmoogra treatment was much more unsatisfactory than at present. First used by Mount about 1854 in the form of powdered seeds, which Travers used as late as 1923 only the oil or some derivative of it is now used. Supposedly but not always, derived from the seeds of *Taraktogenos kurzii*, as obtainable in the earlier days it was usually so rancid that it could be given only by mouth and few patients could stand for any length of time the nausea and irritation caused by the large and frequent doses that were required to obtain any benefit whatever. It appears that Tourtonlis Bey used the oil subcutaneously as early as 1894 but the general adoption of that method was prevented by the irritant qualities of the drug. In an effort to arrive at a method by which it could better be administered parenterally Mercado in Manila devised a formula that could be given intramuscularly to patients of sufficient fortitude. Results were encouraging and Heiser's reports of them led to further experimentation.

With the chemically similar oil of *Hydnocarpus wightiana* Rogers had a sodium soap prepared and in 1916-1917 reported such extraordinarily good results that interest was revived. Shortly afterward, the Hawaiian workers mentioned reintroduced the ethyl esters of chaulmoogra which had been put out commercially at a high price some years before but never adequately tested. In the Philippines, a comparative trial of the Mercado mixture and the sodium salt indicated the latter to be preferable but shortly afterward the ethyl esters,

dilution (though nodules may be touched up with as strong as 50 per cent) applied until there is moderate whitening to an area of 2 to 3 square inches according to the tolerance of the patient, the same area not to be repainted for a month. Paldrock has insisted for years on the value of freezing lesions with solid carbon dioxide and giving complementary treatment with a gold compound. Th freezing is not supposed to serve merely as a counterirritant, but to release antigenic substances and thus lead to immunization.

Potassium iodide was used many years ago for the possible effect of the iodine element, but Danielsen soon found it to be a dangerous drug because of the reactions which it causes and discarded it except as a test for cure. Muir reintroduced it for the express purpose of producing reaction, in the expectation of spontaneous improvement afterward. The unfavorable effects of severe reactions have led to its general condemnation and Muir agrees that it should be used only in suitable cases, under controlled conditions and by experienced persons. The Cairo Committee gave emphasis to the frequent disastrous results that follow its use. In this connection it is of interest that tuberculin was once tried out in leprosy therapy but found only to cause harmful reactions. The induction of simple, nonreactions fever by various means has been tried experimentally without much to encourage the use of such methods. Certain sulfonamides (promin by daily injection, diason and promisol orally) have recently proved beneficial in experimental treatments. They seem likely to replace all other forms of systemic therapy.

A few dyes in therapy have received much attention in recent years, beginning with a preliminary report by Ryne.

Montel has written abundantly on the value of methylene blue, at least as an adjuvant. However after wide trial, the hopes that for a time were held for dye therapy have been largely abandoned, except in connection with lepra reaction.

Treatment of Secondary Conditions.
Lepra Reaction Of the special features of leprosy that call for particular treatment, lepra reaction is outstanding. This condition, often mild and sometimes beneficial, may be very troublesome and quite refractory. Besides general symptomatic treatment, dietetic care, and attention to any discernible condition that may have induced it, various special remedies have been advocated for it and some are of value, but nothing is universally effective in controlling it. Sodium bicarbonate in sufficient amount to make and keep the urine alkaline has often been found helpful. Calcium lactate 1 to 2 gm daily has been used. Calcium chloride intravenously (20 cc. of a 5 per cent solution) has been advocated, but it was not mentioned by the Cairo Committee. In connection with his potassium iodide therapy Muir advocated antimony potassium tartrate for reactions (0.02 to 0.04 gm. intravenously every second day). A less toxic antimony compound, fusidin has been employed with favorable results by Germond, who has recently reported protocols to be effective. Dennev et al found mercurochroms given intravenously to be useful, and Ryne seems to get consistently good results from fluorescein, and the Cairo Committee mentioned both of these substances, as well as potassium antimony tartrate as having proved in different centers to be of value in selected cases.

With regard to chemotherapy treatment of a case in which lepra reaction has appeared, it is a rule that it should be discontinued in the lepromatous form

concentrated and prolonged. A thick lepromatous infiltration so treated once by widely spaced injections may after a time show pits due to decrease of the leproma at the points of injection. With the leprids the active marginal zones are given particular attention but injection of the pale central areas is useful both because there may be latent infection there and because it stimulates repigmentation.

dosage With regard to dosage and frequency of chaulmoogra treatment as a whole practice varies so much that neither the Memorial Conference nor the Cairo Congress Committee made any attempt to lay down definite instructions. Much depends upon the type of the case and also the condition and peculiarities of the individual wherefore, treatment should be individualized. It is a fact that in some countries the average case can be treated more vigorously than in others. For physicians in the Philippines without special experience, Rodriguez advised that the (weekly) dosage starting small should be brought to the totals shown in the following tabulation so long as the patients do well.

No.	Iodine Liqd Esters (cc)	Purified Chaulmoogra Oil (cc)
T 5 cases	1	1 2
From 5 to 6 cases	2	2 3
From 7 to 15 cases	3	3 5
Over 15 cases	4 6	5 10

In some institutions, 5 cc once a week would be considered rather intensive treatment for lepromatous cases and where treatment has to be given on a mass basis the tendency is to keep the dose low in order to minimize disturb-

ances. Neural cases can be given relatively large amounts of the drug and that is particularly important in those of the tuberculoid varieties. With the lepromatous ones, treatment must be more cautious, to avoid reaction and other disturbances, but even so the amount of drug can with many patients, be increased considerably above the amounts ordinarily given. Patients complaints must, of course be given due attention to avoid unnecessary trouble.

The preparation of the drug that is used is one of the controlling factors. A few years ago one institution began to press the dosage of esters severely giving as much as 30 cc twice a week of the esters there available, but after a few months it became necessary to go back to ordinary dosage. On the other hand in Brazil the especially bland esters made there are being used in large doses with corresponding benefit and, it is claimed without ill effects. The Cairo Committee emphasized the importance of the quality of the drug with respect to irritation pointed out, quite reasonably that benefit is directly proportional to the intensity of treatment, and stated that doses "up to 1 cc per 10 pounds of body weight, and even more twice weekly have been used and recommended," and that 30 cc. or more of suitably prepared esters or oil may be tolerated with corresponding benefit.

Other Means of Treatment Mention only can be made of certain other treatment methods, of limited use or usefulness, a full list of things that have been tried would include a very large part of the medical armamentarium. Counter irritation not mentioned by the Cairo Committee is sometimes advocated and has been stressed by Muir he uses trichloroacetic acid chiefly in a 33 per cent

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of the disease. On the other hand Rytie finds that it should be pushed in tuberculoid reaction to double or treble the original dose. With intensive treatment of that kind he prevents or aborts the acute ulcerative tuberculoid condition or if developed it can be caused to heal up rapidly whereas otherwise it may be very protracted.

Other Secondary Conditions Other conditions of this nature can be mentioned only briefly. For *painful neuritis* a manifestation of lepra reaction that is often extremely troublesome and may precipitate trophic changes, counterirritation and heat applied locally or by diathermy have been used. Intramuscular injections of adrenalin have been recommended or ephedrine by mouth also perineural injection of either. Surgical incision or even removal of the capsule (epineurium) to relieve pressure has often been employed. Nerve abscess is similarly dealt with.

Of the *ulcerative conditions* those of leprotic nature that occur late in the disease are typically persistent and resistant to treatment. Dressings of basic fuchsin (1:2000 to 1:1000) have long been used in the Philippines; mercurochrome, acriflavine or potassium permanganate have been employed in the same dilutions. Application of oils rich in vitamin A is said often to cause remarkable improvement, and excellent results with a product called "soluseptamine" given by the intra-arterial (intra-femoral) route have been reported. Persistent ulcerations of the nasal mucosa require special care. Trophic ulcers are notoriously stubborn and difficult to deal with. Protection and cleanliness stand first while various antiseptic and healing substances like those mentioned above may be applied. Infiltration of the tissue around the ulcer or around

the defective nerve, with chaulmoogra drugs has been advocated. Periaxonal sympathectomy has been tried, but the healing that follows it has usually been only temporary the tissue breaking down again within a few months. If necrotic bone is present it must be removed before permanent healing can ever be hoped for. Other conditions, as those of the eye, nose or throat require special attention.

Results of Treatment There are too many variables to permit making any quantitative statement of results obtained in any particular place from the special treatment of leprosy. For success emphasis must be given first to the importance of getting cases early and second to intelligent individualization of the cases and due attention to secondary conditions. There is no way of evaluating precisely the results of treatment. Improvement is slow at best, most striking on the whole in the major tuberculoid cases, which respond comparatively readily to treatment and even subside spontaneously. Ultimate clearing up of the condition is usually uncertain and too often what is accomplished is simply postponement of the unhappy end. Yet, despite all the difficulties, surprisingly good results may be obtained in understaffed and simply equipped institutions where the treatment work is routine and conservative. On the other hand—for reasons not apparent—most discouraging results are reported from better equipped and managed places. There is special concern for the lepromatous type case for even when the patient is restored to normal life the danger of relapse is always present. That is very frequent perhaps in not less than 50 per cent, but it may be delayed for years. The new sulfonamides may be the answer to the problem.

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manifestations in all forms and in all stages (including aleukemic stages) of leukemia, as well as in pseudoleukemia, or Hodgkin's disease and lymphosarcoma. These disturbances may be as follows: (1) Noncharacteristic, or banal, presumably of toxic origin and often called leukemids of Andrey. There may be pruritus of varying severity which may be localized or generalized and the only manifestation, or there may be prurigo-like acroform, urticarial, or bal-

merous mitoses. Large tumors undergo necrosis, nodules rarely.

Without the sternal puncture and the characteristic blood, lymph node and other findings, it is often impossible to diagnose or type the leukemia. In many erythrodermas or cases of exfoliative dermatitis, one finds marked lymphadenopathy commonly not leukemic, but time as well as histologic study may be required to differentiate them from leukemic states. Repeated studies of the



Fig. 328 Leukemia Cutis.

lous lesions, eczematous areas, or ecchymoses, especially in children. (2) They may be characteristic and specific, consisting of (a) diffuse or localized infiltration, which may or may not be part of an eczematous eruption, (b) a generalized leukemic erythroderma (perniciosis lymphoderma of Kaposi) and (c) slow-growing leukemic tumors varying in number and size from small subcutaneous and cutaneous pea-sized, skin-colored, or bluish-red nodules to rather large masses. The mucosae, especially the gums, may be involved, the latter are apt to show histologically dense, dermal, lymphocytic infiltrates, often with nu-

merous mitoses. Large tumors undergo necrosis, nodules rarely.

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Spliegler Fendt Sarcoid

This misnomer has been given to a lymphoblastomatous manifestation characterized by millet-seed to pea and larger-sized, red to brownish, firm but not hard, few or many well-defined papules and nodules. The surface of these lesions is apt to show telangiectases. They do not ulcerate. The lesions often resemble sarcoma cutis, carcinomatous cutis, and metastatic melanoma. Diagnosis must

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LEUKEMIA CUTIS

SYNONYM *Leukocythemia.*

Leukemia is characterized by a hyperplasia of the leukocyte-producing tissues usually associated with marked increase in the leukocytes of the circulating blood

Incidence Males are affected more frequently than females and it is more prevalent toward middle life

Etiology The cause is not known

Varieties There are three varieties of leukemia the myelogenous the lym-

phatic and the monocytic The monocytic variety is divided into two types the Naegeli and Schilling types. Myelogenous and lymphatic leukemia may be either acute or chronic while monocytic leukemia is always acute

Cutaneous Lesions

A number of cutaneous disturbances may precede or accompany systemic

ination of the blood shows an increase in leukocytes; however the count does not often exceed 100,000 and about 80 per cent of these are small lymphocytes.

Symptoms. Pallor is an early symptom and, as the disease progresses, loss of weight and edema of the extremities ensue. Fever occurs late. Pruritus and exematoid plaques are commonly present. Cachexia is the usual cause of death which most frequently occurs from three to six years of age.

Acute Myeloblastic and Lymphatic Leukemia

These are clinically similar. They occur more frequently before the twentieth year. Red blood cells in a few weeks drop from normal to 3,000,000 and the hemoglobin to 25 per cent. There is a thrombocytopenia. The leukocytes range between 10,000 and 100,000. The acute variety is characterized by a purpuric and bullous eruption, rapid pulse, palpable spleen, and rapid enlargement of the lymph glands of the neck and jaw.

Monocytic Leukemia

This occurs only in the acute form and is rapidly fatal. This type occurs as either Schilling or Naegeli type. The acute Schilling type is characterized by the presence of a great majority of leukocytes showing a tendency to develop into monocytes. The Naegeli type of monocytic leukemia is a variant of myelogenous leukemia in which it often terminates. The onset of monocytic leukemia is not unlike other leukemias except that it is more acute and ulceration of the mouth and pharynx commonly occurs, sometimes going on to gangrene. Lymph nodes and the spleen are only slightly enlarged. Purpuric,

hemorrhagic, and bullous lesions frequently develop early in the course of monocytic leukemia. Exfoliative dermatitis and erythrodermia are types of specific cutaneous reactions which commonly occur.

In both the myeloid and lymphatic forms of chronic or acute leukemia, there



Fig. 331. Lymphatic Leukemia. (Courtesy of Dr. Carroll S. Wright.)

are periods when the blood picture is entirely normal. This stage is referred to as aleukemic leukemia.

Treatment

There is no specific therapy for any form of leukemia. Ethyl carbamate (urethane) 1 gm. (15 grains) three times daily has been used with benefit in leukemia, especially myeloid types. Nitrogen mustard gas and parabenzquinone have been used to some avail. Roentgen irradiation, generalized ultraviolet radiation, arsenic, blood transfusions, benzol, and fever therapy have been used with varying results.

be made histologically there is found more or less circumscribed, round-cell infiltration of the cutis, rarely of the epidermis and papillary layer

Chronic Myeloid Leukemia

This is a hyperplasia of the cells of the bone marrow



Fig. 329: Leukemia Cutis. Note nodular subcutaneous infiltration on forehead of three years duration.

Pathology The spleen is greatly enlarged. The lymph nodes are only slightly enlarged if at all. Ascites may be present. The most diagnostic feature is revealed by the blood examination. The number of red cells is usually reduced and may be only two or three million. Poikilocytosis as well as anisocytosis may be present. Normoblasts and in severe cases megaloblasts may be present. The most striking feature is the increase in leukocytes, a leukocyte count of 400,000 and more is not uncommon.

Symptoms The initial symptoms are those of asthenia and gastrointestinal disturbance while hemorrhage from the nose and gums, hemorrhage retinitis, and hemorrhage in the internal ear are not uncommon. Pain over the bones is also characteristic. This is a fatal disease; remissions do however occur and the disease terminates in from two to five years.

Chronic Lymphatic Leukemia

SYNONYMS: *Lymphoid leukemia.*

This variety is a hyperplasia of the lymphatic tissue.

Incidence It occurs later in life than the myelogenous type, more often between fifty and sixty. Males are also more frequently affected.



Fig. 330 Lymphoblastoma.

Pathology The lymph nodes are enlarged but they do not mat together. Lymph nodes of the chest, abdomen, and throat are also involved. The spleen is enlarged however to a lesser degree than in the myelogenous variety. Exam-

gauge needle attached to a tuberculin syringe. Two to three drops of the alcohol are injected just below the derm in each square centimeter of thickened skin. Treatment may be repeated at five- to seven-day intervals.

Recurrences of lichen simplex chronicus are frequent even after the most careful treatment. The following anti-pruritic preparations are suggested for use following radiotherapy:

I	
Liq. carbonis detergens	20
Piccol	75
Glycerin	80
Menthol	05
Spts. cal rect.	800
Aqua camphor	1000

See For use on the body and the scalp

II

Oil of turpentine or crude coal tar

Use Paint involved areas twice each day

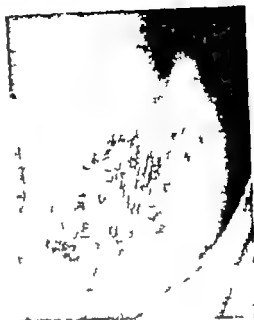


Fig. 333 Lichen Chronicus Simplex (neurodermatitis) Of axilla.

LICHEN PLANUS

SYNONYMS *Lichen ruber planus*, *lichen porphyria*.

Lichen planus is an acute or chronic inflammatory disease of the skin and mucous membranes characterized by glistening and angular papules varying in color from red to violet, which have a predilection for the flexor surfaces of the body.

Varieties The varieties of lichen planus comprise the following eight clinical types: (1) lichen planus annularis; (2) lichen planus linearis; (3) lichen planus trophicus; (4) lichen planus bulbosus; (5) lichen planus hypertrophicus; (6) lichen planus erythematosus; (7) lichen planus haemorrhagicus; and finally (8) lichen-sclerosus et atrophicus (H. I. Koppeau).

Incidence Lichen planus occurs more often after the second decade of life. It is rare in children and occurs equally in both sexes.

Etiology The etiology of lichen planus is unknown. Grief anxiety shock, and mental strain frequently precede the disease. Some authors believe bacteremia, diminished vasomotor tonus, trauma and virus infection are causative agents. Lichen planus, differing clinically and histologically in no way from the spontaneous types, has been observed at times to follow the use of arsenical, bismuth, and gold injections, and the ingestion of atabrine (quinacrine hydrochloride) for malaria.

Pathogenesis Lichen planus papules arise in the true skin as circumscribed areas of round cell infiltration about the dilated capillaries. This band of small round cell infiltration is pathognomonic of lichen planus. The epidermis overlying these areas of infiltration is thickened. The stratum granulosum

LICHENIFICATION

Long-continued irritation of the skin by rubbing and scratching leads to a thickened and leathery appearance with mosaiclike texture to the cutaneous surface. The involved area appears as a flat, shiny, more or less angular and elevated lesion with exaggerated furrows lying between lesions. The area may become markedly papular (giant lichenification). Lichenification may be primary or secondary. Primary lichenification occurs on seemingly normal skin and is known as idiopathic or lichen simplex chronicus. Secondary lichenification develops upon a skin afflicted with some disturbance like eczema, ringworm, etc., and is known as secondary neurodermatitis. (See also Frictional Dermatitis, p 250.)

Lichen Simplex Chronicus

SYNONYMS: *Neurodermit chronique circonscrite (Jacquet)*; *pruritus acrie lichenification (Brocq)*

Lichen simplex chronicus is a form of localized neurodermatitis.

Etiology: Lichen simplex chronicus is ascribed to various etiologic factors among which nervous irritability, lack of emotional control and autointoxication may be mentioned.

Pathology: The histopathology reveals thickening of the epidermis, hyperkeratosis, and prolongation of the interpapillary processes. Focal perivascular cellular infiltration of the papillary zone of the corium consists of lymphocytes, a few fibroblasts and eosinophiles. Slight edema and vascular dilatation are evident.

Symptoms: The outstanding objective symptom is a varying degree of lichenification. The subjective symptom is pruritus. Lesions may at times be de-

cidedly papular resembling lichen planus. They may also appear as excoriated, slightly scaly and moist patches, which are rarely nodular.

The disease has predilection for the back and sides of the neck, the antecubital and popliteal areas.

Treatment: The purpose of treatment is to control the pruritus. This is



Fig. 332. Giant Lichenification. Of scrotum. (Courtesy of Dr. F. Roese.)

best accomplished by roentgen ray irradiation. Sedatives and antipruritic remedies are employed. Wet dressings and medicated baths are helpful. Residual thickenings are ameliorated by keratolytic ointments containing sulfur and salicylic acid. Autohemotherapy is occasionally beneficial. An occlusive dressing using elastoplast, whenever possible and applied for one week at a time, may help.

In resistant cases, Pels and Ellis have obtained good results with injections of 95 per cent alcohol using a 25- to 27



Fig. 335 Lichen Planus.

is markedly hypertrophied. The lesions are traversed by ducts of the sweat glands.

Symptoms. The subjective symptom of lichen planus is itching which is often intolerable and paroxysmal. The acute type is usually generalized while



Fig. 331: Lichen Planus. Close view; note the numerous small papules and the lichenified areas largely due to rubbing. (Courtesy of Dr. Jacques P. Guequiere.)

the chronic type is localized. In the chronic type itching is less severe and it is occasionally absent.

Acute lichen planus develops rapidly so that the entire body may be covered within twenty-four hours. The papules are violaceous in color, small, flat, umbilicated and rest upon rounded angular or polygonal bases. The surface of a lichen planus papule may present a fine whitish network of striae known as Wickham's striae which are capped by closely adherent scales. The arrangement of lichen planus lesions follows the normal

cutaneous lines of cleavage. Coalescence of papules is less frequent in acute lichen planus than in chronic forms. Papules often appear at sites of irritation so that a row of papules may develop over a scratch mark; this feature is known as "Kobner's phenomenon" and is also characteristic of psoriasis.

Chronic lichen planus begins insidiously with symmetrical lesions. The sites of predilection are the wrists, inner aspect of the legs, lumbar spine, scrotum, penis, mucous membrane of the mouth, lips, and vagina. The face and scalp are rarely involved. Healed lesions tend to leave temporary pigmented atrophic scars, especially when the disease is of long duration.

Buccal lesions occur in about 40 per cent of patients with lichen planus. The mucous membrane of the vulva, anus, urethra, and conjunctiva may occasionally be involved. Lesions of the oral mucous membranes are of two forms, namely (1) flat, delicate bluish-white lacelike network covering the mucous membrane of the cheek, and (2) firm, elevated papules which may be discrete or confluent on the tongue. Tongue lesions may coalesce and completely cover the dorsum. Oral lichen planus may antedate cutaneous lesions by many weeks, or may be the only site of involvement.

Vulvar lesions are similar in appearance to mouth lesions except for the presence of atrophy.

Abortive forms are not rare. In these only a few lesions may be present especially on flexor surfaces, and the itching may be generalized or limited to the lesions themselves.

The palms and soles are rarely involved. Palmar and plantar lesions, when present, are hyperkeratotic and leave a cribriform appearance on des-

white patches. It simulates morphea guttata, however the white spots in this disease are oval or round while in lichen planus atrophicus the spots are angular and a central umbilication is often present in them.

Lichen planus bullousus is characterized by the presence of bullae and vesicles



Fig. 837 Lichen Planus. Common location for this disease is the shaft and glans penis. Note the flat, pinhead-sized, polygonal papules. (Courtesy of Dr. Jacques F. Coquerelle)

They are more likely to occur on the extremities. Nikolsky's phenomenon, in which the upper layers of the epidermis are easily removed by slight pressure, may be present.

Lichen planus hypertrophicus (lichen planus verrucosus) is characterized by hypertrophic or verrucous plaques covered with a fine adherent scale. The site of predilection is the anterior aspect of the lower extremities; it may however occur on other parts of the glabrous skin surface. The adherence of the scales,

the color and the itching differentiate it from a patch of psoriasis.

Lichen planus erythematosus is characterized by an erythema in the area in which lichen papules later occur. The erythema is the prodrome of a lichen planus, which may precede it by many months.

In *lichen planus hemorrhagicus* the lesions are hemorrhagic.

Lichen Sclerosus et Atrophicus (Hallopeau) (*Lichen Planus et Acuminatus Atrophicus Folliculitis Decalvans* of *Lichen Sprunlorus* of Little *Lichen Planopilaris* of Pringle) This variety is characterized by the appearance of ivory colored papules which are umbilicated and usually atrophy early in the course of the disease. The site of predilection is the scalp, it is no doubt a syndrome of cicatricial alopecia.

Lichen Sprunlorus and *Pseudopelade* These are frequently associated with and are considered by some as varieties of lichen planus of the scalp.

Diagnosis The disease is differentiated from psoriasis, lichenoid eczema, and papular syphilides. Psoriasis has a predilection for extensor surfaces, the elbows, knees, and scalp. Psoriatic papules are scaly from the beginning and removal of these silvery white scales exposes bleeding areas. There is absence of central umbilication in papules of psoriasis.

Papular eczema does not involve mucous membranes and lacks the distribution of lichen planus. The silverlike stippling of the mucous membrane with stellar and reticular lesions is characteristic of lichen planus and differentiates it from leukoplakia.

Lupus erythematosus is more inflammatory and often erosive. The blood in lupus erythematosus usually shows a leukopenia with lymphocytosis. The ab-

quamation. If the nails are involved the matrix is edematous and the nail itself is grooved and striated.

Characteristics of Clinical Types
The several varieties of lichen planus have certain definite characteristics.

Lichen planus linearis is characterized by bandlike patches which are similar in appearance to lesions of herpes zoster. The site of predilection is the posterior surface of the lower extremities. They often resemble a linear nevus except



Fig. 336. Acute Lichen Planus. Of one week's duration.

In *lichen planus annularis* the papules either coalesce or are arranged in a circinate manner. They may occur anywhere on the body surface; however the sites of predilection are the penis, mesial surface of the thighs, and the mucous membranes.

that in lichen planus the itching is intense.

In *lichen planus atrophicus* the center of the papule undergoes atrophy. Atrophic white spots mark the site of the former papules. Coalescence of these atrophic areas forms glistening ivory or



Fig. 340 Lichen Planus. Involving tongue, inner lip, and buccal.



Fig. 341 Lichen Planus. *Left* Typical, of the buccal mucosa. *Right* Of the palate (erosive type). Note the delicate reticulated white lines radiating from the margin of the lesion.



Fig. 338: Lichen Planus. Sloughing lesions on penis and hand.



Fig. 339 Lichen Planus. Plaque on center of the tongue.



Fig. 243 Lichen Atrophicus.

sence of an inflammatory areola differentiates oral lichen planus from lupus erythematosus of the mucosa.

The positive Wassermann reaction, the absence of itching and the concomitant lymph adenopathy are diagnostic of *sypilis*.

and last for many years. Hypertrophic and verrucous patches, usually present on the legs, are resistant to treatment and usually require years of therapy.

Treatment There is no specific although arsenic, bismuth, and mercury have been employed extensively for treat-



Fig. 342: Hypertrophic Lichen Planus.

Prognosis - Acute lichen planus responds more readily to treatment than the chronic type and the more generalized the eruption the quicker is the response to treatment. Acute eruptions of lichen planus usually run a self-limited course but may become chronic.

ing lichen planus. They are advised when arsenic, bismuth, gold or atabrine are not incriminated as possible etiologic agents. Arsenic may be given by mouth in the form of the Asiatic pill containing arsenic trioxide and black pepper. The pills contain from $\frac{1}{32}$ to $\frac{1}{16}$ grain of arsenic.

LYMPHOGRANULOMA VENEREUM

SYNONYMS *Lymphogranuloma inguinale*, *lymphogranuloma tropicum*, *lymphogranulomatosis inguinale*, *Nicolaï-Ferre-Durand disease*, *poradenitis*, *lymphopathia venereum*, *tr. pica* or *climatic bubo*.

Lymphogranuloma venereum is a venereal disease characterized by a small initial lesion followed by subacute regional lymphadenitis, and accompanied by constitutional symptoms.

Varieties Varieties of lymphogranuloma venereum are classified according

to Larström and Wassén. It grows luxuriantly in the yolk sac of the chick embryo.

Pathology The primary lesion is an inflammatory nodule which undergoes necrosis. The necrotic area is composed of cellular debris of leukocytes and lymphocytes surrounded by epithelioid cells.



Fig. 345 *Lymphogranuloma Venereum*. Acute or early unruptured lymphadenopathy

to location and they are either inguinal, genital, anal, rectal, urethral, or extra-genital.

Incidence: *Lymphogranuloma venereum* is about one tenth as common as chancroid. It is more often seen in the Negro race, and in the southern states and eastern seaboard. Males and females are equally affected.

Etiology The causative agent is a filterable virus, originally isolated by Hel

The histopathology is that of an infectious granuloma and the formation of star-shaped abscesses. The blood picture reveals an increase in monocytes and eosinophiles. The histological picture is not in itself diagnostic.

Symptoms *Lymphogranuloma venereum* is a venereal disease transmitted by sexual intercourse but children may contract the infection by contact with douche nozzles, infected clothing, etc.

trioxide. It is given one hour after each meal, increasing the dose to the point of tolerance. Arsenic may also be administered subcutaneously. Stovarsol is a reliable arsenical preparation to employ. It may be given intravenously at weekly intervals in the form of sodium cacodylate (0.5 gm [7.5 grains]).



Fig 314 Lichen Atrophicus.

Bismuth is given intramuscularly in the form of bismuth subsalicylate in oil in doses of 2 cc each week for a period of ten weeks. This course of treatment may be repeated after a rest of one month.

Cyanide of mercury is given intravenously every third day in doses of 0.01 gm ($\frac{1}{10}$ grain) for one month and has proved to be a worthwhile mode of treatment. In the acute type, intramuscular injections of a 1 per cent solution of bichloride of mercury in isotonic salt solution (1 cc twice weekly) is definitely helpful. In some cases subcutaneous injections of *liver extract* have also afforded excellent results.

The pruritus, in both acute and chronic lichen planus, can often be controlled by

a three to five-day diet limited to rice or buttermilk.

Generalized ultraviolet radiation and sedation by intravenous injections of strontium bromide (10 cc. of a 10 per cent solution every other day) or oral phenobarbital (gr as four times a day) are helpful in some cases, especially in those visibly under mental strain.

In localized areas, roentgenotherapy is useful in relieving the accompanying pruritus. Filtered roentgen rays over the spine is a valuable adjuvant to treatment for generalized types.

For the treatment of lesions of the mucous membrane, *bismarsen* and *enesol* are recommended. *Riboflavin* and *nicotinic acid* should be given in all cases of mucous-membrane involvement.

The following local remedies are used to combat pruritus.

Phenolis	40
Acidi borici	40
Sulfur colloidal	80
Resorcinolis	20
Lq camphorae q s ad	1200

Sol. Shake well and apply with a small pad. Dilute in water if too strong.

Tar ointment and *liquor carbonis detergens* are useful for treating thickened hypertrophic forms of lichen planus.

Acidi salicylici	10
Mentholis	0.15
Crude coal tar	2.0
Emplast. plumbi and petrolatum q s ad	300

Sol. Apply t. 4 or 5 times each day. Pad cover treated areas with light dressing.

In the hypertrophic type, the following powder has proved satisfactory.

Mentholis	0.2
Acidi salicylici	30.0
Acidi borici	60.0

Sol. Apply olive oil to lesion, then apply this powder.

Solid carbon dioxide snow applications and strong reducing agents, such as *chrysarobin* (1 to 5 per cent) or *neorobin* ($\frac{1}{4}$ to 1 per cent) may also be used.

perglobulinemia in thirty-eight of forty two cases with a tendency for the results of the test to return to normal after clinical cure.

A latent infection is more common than is generally supposed as based on positive Frei and complement fixation reactions.

Diagnosis Diagnosis is established by the following measures.

Frei Test The most important and

confirmatory test, according to Robert H. Greenblatt, is the Frei test. The most reliable preparations are antigens of infected yolk of chick embryos with a control material from uninfected yolk. The test is performed by injecting 0.1 cc. of the antigen intradermally in the forearm. In positive cases, an inflammatory infiltration of nodule measuring 3

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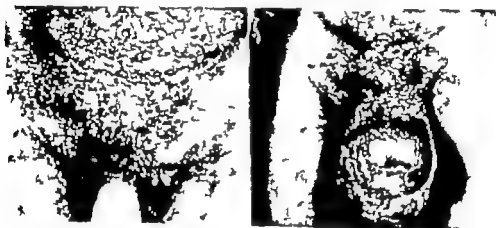


Fig. 347 Lymphogranuloma Venereum.

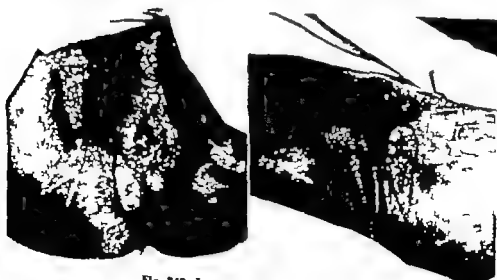


Fig. 348 Lymphogranuloma Venereum.

Lymphogranuloma Venereum

The incubation period varies from four days to four weeks. The inguinal bubo usually appears from ten to thirty days after exposure. The primary lesion appears as a slightly infiltrated papule, a small erosion or a vesicle resembling herpes. The primary lesion appears more often on the glans penis or prepuce in males, and on the cervix, posterior fornix labia or clitoris in females. These lesions are evanescent manifestations of the infection which lasts for several

tent, remittent or continuous. The picture of lymph node involvement in lymphogranuloma venereum is different among females because the lymph channels draining the vulva communicate with the lymph nodes surrounding the lower part of the rectum. This leads to involvement of the rectal wall which often results in anal stricture of the lower rectal wall. Rectal fistula may arise in females in consequence of proliferation about the anus. Chronic hy-



Fig. 346: Lymphogranuloma Venereum. *Left:* External involvement of lymphatics of groin and lower abdomen with numerous fistulous tracts. *Right:* Positive Frei. Note multiple discharging sinuses of left groin and inguinal region.

weeks. The regional inguinal nodes of one or both sides become enlarged. Unilateral lymph node involvement is the usual result. The adenitis in lymphogranuloma venereum is characteristic in that the regional lymph nodes fuse together to form a large mass half the size of a fist. The cutaneous surface over areas of adenitis assumes violaceous color. Breaking down of the enlarged lymph nodes leads to multiple fistulas. The accompanying constitutional symptoms consist of malaise, fever, loss of appetite, loss of weight, periarticular pain and polyarthritides and cutaneous eruptions. The fever may be intermit-

tent, remittent or continuous. The picture of lymph node involvement in lymphogranuloma venereum is different among females because the lymph channels draining the vulva communicate with the lymph nodes surrounding the lower part of the rectum. This leads to involvement of the rectal wall which often results in anal stricture of the lower rectal wall. Rectal fistula may arise in females in consequence of proliferation about the anus. Chronic hy-

peritrophic vulvitis and elephantiasis of scrotum and penis arise. A variety of allergic eruptions simulating erythema multiforme and erythema nodosum have been observed to accompany lymphogranuloma venereum. Acute and chronic polyarthritides and arthralgias have likewise occurred. Extragenital infection with ulceration of the tongue and cervical lymphadenitis have been described by Curth, who also called attention to ulceration of the colon, and to proctitis. Numerous cases of extragenital infection have since been reported. Combes and his co-workers found a positive formaldehyde-gel test and a hy-

perglobulinemia in thirty-eight of forty-two cases with a tendency for the results of the test to return to normal after clinical cure.

A latent infection is more common than is generally supposed as based on positive Frei and complement-fixation reactions.

Diagnosis. Diagnosis is established by the following measures:

Frei Test. The most important and

confirmatory test according to Robert B. Greenblatt, is the Frei test. The most reliable preparations are antigens of infected yolk of chick embryos with a control material from uninfected yolk. The test is performed by injecting 0.1 cc of the antigen intradermally in the forearm. In positive cases, an inflammatory infiltration of nodule measuring 3

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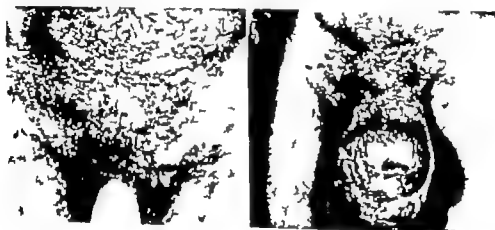


Fig. 347 Lymphogranuloma Venereum.

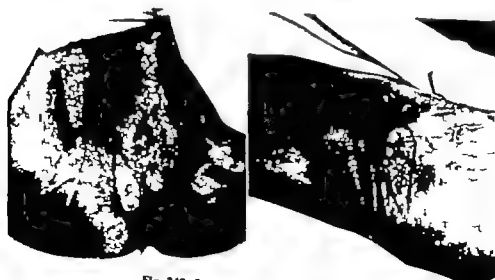


Fig. 348 Lymphogranuloma Venereum.

to 6 mm in diameter with a surrounding zone of inflammation occurring at the end of forty-eight to seventy-two hours. If the test is negative the same area should be examined at intervals for several days so as to discover delayed reaction which occasionally occurs. A



Fig. 319: Lymphogranuloma Venereum. Secondary pseudoelephantiasis.

small area of necrosis or the occurrence of a bleb in the center of the nodule or indurated zone is diagnostic of a strongly positive reaction. If the reaction is negative it should be repeated. The control test reaction when uninfected chick embryo material is used should measure not more than 3 mm. Twelve to forty or more days may elapse from the time of a primary lesion before a positive Frei test is obtained. A positive Frei test continues during the life of the patient unless the patient becomes so

debilitated that a state of anergia exists when a negative test results. Where early sulfonamide therapy is employed, a positive test may become negative; however when the disease has already been well established, the test remains positive despite all therapy.

Biopsy In the benign type of lymphogranuloma venereum, biopsy will differentiate malignancy and Hodgkin's disease. In many ulcerative lesions, biopsy offers a means of diagnosis, particularly where there is a positive reaction to serologic tests for syphilis; however the histologic picture of lymphogranuloma venereum is only suggestive and not necessarily specific.

Autoinoculation. Autoinoculation of aspirated pus from a bubo of lymphogranuloma venereum does not produce a local ulceration, while in chancroid the inoculation of a pirated pus frequently produces a chancroidal ulcer.

Complement Fixation Test. A serologic complement fixation test with antigens from lymphogranuloma venereum virus of chick embryo origin is available. It is of much value in early diagnosis and as a gauge of the progress of the disease.

Inverted Frei Test. A negative Frei test may occur even in the presence of a suppurative bubo. In this event the pus aspirated from such a bubo may be made into an antigen and on injection into known cases of lymphogranuloma venereum will produce a positive reaction. Patients with positive reactions to serologic tests for syphilis, positive chancroid skin test or fistulating bubo should not be used.

Differential Diagnosis. Lymphogranuloma venereum must be differentiated from granuloma inguinale, syphilis, gonorrhea, herpes progenitalis, fusosporochetosis, blastomycosis, benign tumor and malignancy.

Granuloma inguinale is diagnosed only when Donovan bodies are found or when the histologic picture reveals the pathognomonic cells. Syphilis is diagnosed by dark field examination for *Sporochæta pallida* and a positive reaction to serologic tests for syphilis. Gonorrhea is diagnosed by the finding of the gonococcus. Herpes progenitalis is diagnosed by the multiple lesions and a history of previous involvement. *Fusosporochetosis* is diagnosed by finding of spirillum and vibrio of Vincent. Blastomycosis is diagnosed by the presence of blastomyces. Malignancy and benign tumors are differentiated from lymphogranuloma venereum by histological examination.

Complications. Malignant manifestations of the anus or vulva may complicate lymphogranuloma venereum. Inguinal bubo with vulvar elephantiasis, intestinal strictures which often lead to intestinal obstruction, and urethral strictures are not unusual complications.

The disease may be present for years and may be so debilitating as to produce a life of invalidism. Elephantiasis of the vulva and genitalia occurs most frequently in the Negro race.

Prognosis. Many therapeutic measures have been employed in the treat-

ment of lymphogranuloma venereum. In the majority of cases, the results are not satisfactory.

Treatment. Successful treatment depends upon early diagnosis and prompt institution of effective therapy. Injections of Frei antigen are given once a week over a period of months, starting with 0.1 cc. and gradually increasing the dose to 2 cc. or it may be given intravenously once a week in doses of 0.05 to 0.5 cc. Febrile reactions may accompany early injections.

Röntgenotherapy is beneficial in early cases of infection, while in advanced cases free drainage is indicated.

Nonspecific protein injections have considerable value. Gold and iodide therapy also have their advocates.

Sulfa ilamide is perhaps the treatment of choice, and in this disease is certainly virustatic if not virucidal. The dose is 2 gm., given three times each day for fifteen days, followed by a period of rest covering ten days. Sulfanilamide therapy is then repeated. This type of therapy if combined with Frei antigen injections, gives excellent results.

Conalescent serum or transfusion of blood from patients who have had lymphogranuloma venereum has also proved of value.

MALUM PERFORANS

SYNONYMS: *Malum perforans pedis* perforating ulcer *perfori endes fuugschwur*
mal perforant du pied

Malum perforans is a chronic trophic perforating ulcer usually occurring on the plantar surface of the foot.

Incidence It is seen more often among males between twenty and fifty years of age who are suffering from locomotor ataxia, chronic nephritis, diabetes mellitus, arterio-sclerosis, and leprosy.

Symptoms The condition begins as a localized callosity (not unlike a corn) on the plantar surface of the foot, usually situated over the articulation of the metatarsal bone with the phalanx of the first or last toe. These areas are subjected to the most pressure and trauma.



Fig. 350. *Malum Perforans* (perforating ulcer). A probe could be passed painlessly into this ulcer for almost 10 mm.

Etiology The primary cause is injury to a nerve trunk or nerve center and nerve degeneration. The underlying cause in the majority of cases is unknown. Some of the cases are attributable to syphilis, malaria, rheumatism, and traumatism.

Pathology Neuritis and thickening of the neurolemma is present in the nerves distributed to the affected parts. Sclerosis of the posterior cornua and columns of the cord has also been demonstrated.



Fig. 351: *Malum Perforans*. A post-encephalitic trophic ulcer of four years' duration. It was completely anesthetic.

Beneath this corallike area, inflammation and suppuration take place and the overlying horny covering is thrown off disclosing a shallow ulcer. This ulcer gradually extends to the bone, which also becomes involved in this necrotic process. There is little discharge from this sinus and anesthesia of the area involved is the rule. Pain and tenderness, if present at all, is slight. The ulcer is usually single; however several may occur and occasionally both feet are affected. The palm, nose, and mouth are occasionally the sites of perforating ulcers. Hyperhidrosis, callouses, and

nail changes are associated symptoms.

Diagnosis This must be differentiated from syphilitic and tuberculous ulcers, leprosy and diabetic gangrene. A mistake in diagnosis is rarely made.

Prognosis The disease is chronic and lasts indefinitely.

Treatment: Malum perforans is resistant to local therapy. Roentgenotherapy may however be beneficial and is worthy of trial. An underlying diabetes requires insulin. Rest, removal of necrotic tissue and hot boric acid compresses are helpful. Digital amputation may be necessary.

MILIARIA

SYNONYMS *Prickly heat, heat rash, lichen tropicus, milium impetigo (Darier)*

Miliaria is an acute, mildly inflammatory eruption, characterized by pinpoint to pinhead-sized red discrete papules, papulovesicles, and vesicles, often accompanied by itching and burning and usually developing on parts subjected to excessive perspiration.

Etiology The disease is commonest in hot weather in the obese and in the debilitated, as well as in perfectly healthy infants, children, and young adults. Sweating precedes or accompanies the eruption. Epidermis maceration appears to be predisposing. It is common in those who perspire easily in those who are overclothed, exercise strenuously work in moist or excessive heat, and after a bout of fever. Preexisting skin infections—impetigo and furunculosis—favor its development. Smith isolated a yeast like fungus in material removed from the lesions.

Pathology Histologically the lesion may be found to be a minute bulla due to separation of the stratum corneum or vesicle the result of real spongiosis. It is often a combination of both processes.

The center of the lesion is often marked by a sweat gland orifice" (Darier).

Symptoms Miliaria develops more or less overnight, and is prolonged by the condition which favored its original development, under which circumstances new crops of lesions appear.

The lesions are at first pinhead-sized red spots, centered by a minute vesicle with cloudy contents, which on culture shows low virulent cutaneous staphylococci (micrococci of Unna). Elevation of the lesion is apparent but not distinctly palpable; there is no induration and no oozing. The sites of predilection are the trunk, the arms, and thighs. In most cases one finds papules and papulovesicles. Sometimes the entire eruption is composed of minute papules; in others, vesicles with pink or red areolas comprise most of the eruption. The lesions are discrete and closely placed, but the skin as a whole is hyperemic. The subjective symptoms vary from slight to severe degrees of itching and burning.

Diagnosis A history of marked sweating and overheating of the body

followed by the acute onset of minute, closely placed mildly inflammatory lesions, is characteristic. Typical papular and papulovesicular *eczema* is accompanied by oozing is more inflammatory but less extensive, occurs in patches does not develop as suddenly and is more persistent. In *sudamina* there are no signs of inflammation.

Prognosis Properly treated miliana runs a course of four or five days. Recurrent attacks may prolong it for several or more weeks.

Treatment Avoidance of excessive heat and clothing are preventive measures. In persistent cases, frequent *ultra violet radiation* is beneficial. The dusting powder or lotion noted below may be used prophylactically. Frequent washing with 70 per cent *alcohol* or with a 1:2400 solution of *bichloride of mercury* and a simple dusting powder such as

Menthol	0.5
Salicylic acid.	2.0
Boric acid	50.0
Talc	100.0

is sufficient for the actual condition. The following *lotions* in itching or burning are of value

I	
Phenol	5.0
Glycerin	100
Boric acid	5.0
Magma bentonite (N F)	50.0
Water q. s. ad.	100.0

II	
Precipitated zinc carbonate	50.0
Witchhazel water	50.0
Cresote	5.0
Lime water q. s. ad.	100.0

III	
Menthol.	1.0
Zinc carb. precip.	25.0
Sp. vin. rect. (70%) q. s. d.	250.0
M	

MILIUM

SYNONYMS: *Acne albidus*, *staphylus albidus*, *granum acn miliar tuberculi* in *sebaceum*, *pearly tubercles*, *white heads*.

A milium is a small, opaque white or yellow sharply circumscribed rounded noninflammatory cyst situated in the upper part of the corium.

Varieties MacLeod identifies three types of milia.

1 Small round cystic lesions which contain sebaceous material and epidermal debris. These lesions are seen frequently on the face and genitalia (*staphylus albidus*) of infants.

2 Small opaque encapsulated masses which occur in association with rodent ulcer, benign cystic epitheliomas, wounds, following exfoliation in pemphigus, epidermolysis bullosa, dermatitis herpetiformis, and scarlatina.

3 True milia, in which the lesions are small, round slightly compressed and

contain inspissated fatty sebaceous material. The contents of a milium may occasionally undergo calcification. Milia may be congenital.

Incidence Milia occur in both sexes and at any age from infancy on to advanced life however they are more common in adult life. They are frequently seen on the eyelids and malar regions of women past middle life, particularly among those with seborrhea. The condition may be congenital or acquired.

Etiology Milia may be produced accidentally or they may be of embryonic origin. They often complicate syphilis, tuberculosis, pemphigus, erysipelas, epidermolysis bullosa and other vesicular eruptions.



Fig. 353 Miliaria. (Courtesy of Dr. Carroll S. Wright.)

followed by the acute onset of minute closely placed mildly inflammatory lesions is characteristic. Typical papular and papulovesicular *eczema* is accompanied by oozing is more inflammatory but less extensive occurs in patches, does not develop as suddenly and is more persistent. In *sudamina* there are no signs of inflammation.

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Glycerin	1.00
Boric acid	5.0
Alum bentonate (N F)	60.0
Water q s ad	100.0

II	
Precipitated zinc carbonate	40.0
Witchhazel water	32.0
Crotonole	0.9
Lime water q s ad	100.0

III	
Menthol	1.0
Zinc carb. precip.	25.0
Sp. vini. rect. (70%) q s. ad.	100.0
U	

MILIUM

SYNONYMS: *Acne alba* et *ophius albidus*, *gratum*, *acne miliare*, *tuberculum sebaceum*, *pearly tubercles*, *whiteheads*.

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Etiology Milia may be produced accidentally or they may be of embryonic origin. They often complicate syphilis, tuberculosis, pemphigus, erysipelas, epidermolysis bullosa and other vesicular eruptions.

Pathology Milia probably represent a retention product of the sebaceous glands. Others (Virchow) believe them to result from a hyperkeratosis of the epithelium of the hair follicles. Roberson believes that milia originate from embryonic epithelium of hair follicles.

Symptoms Milia occur on the face, genitalia, and other parts of the integument. The sites of predilection are the eyelids and malar regions. The lesions vary in size from a pinpoint to a pin head. They are solid elevations which are usually discrete and irregularly scattered. Several lesions may be grouped to form a coalescence. Milia have no symptoms. They are disfiguring but harmless. Lesions very rarely undergo calcareous metamorphosis to form stony like projections from the skin. A milium is insidious in onset, has a tendency to grow slowly and may persist indefinitely because it does not undergo spontaneous involution.

Diagnosis Milium is distinguished from comedo by the absence of the central black plug and the duct orifice present in a comedo. *Molluscum contagiosum* is identified by the characteristic umbilication, its large size, and rapid growth. Xanthoma is ruled out by the appearance of soft, wrinkled, chamous-colored plaques.

Treatment The treatment of choice is rasuion with a very fine, sharp iridec

tomy knife and excudation of the cystic material. Application of a 50 per cent solution of *chromic acid* prevents recurrences. The occasional use of mechanical *keratolytics* such as pumice stone has been advised. *Electrolysis* and the



Fig. 353 Milium. Single large lesion such as this is unusual; milia are usually multiple, pinpoint to pinhead, firm, whit elevations.

for *galvanocautery* are the most convenient methods of removal.

An ointment containing 5 per cent salicylic acid and 10 per cent sulfur may also be used. Peeling with the cold quartz light is useful.

MOLLUSCUM CONTAGIOSUM

SYNONYMS *Molluscum pitelliale*, *pitelloma contagiosum*, *scus varioliforme* (Boris), *molluscum scaberrimum*.

Molluscum contagiosum is a contagious, autoinoculable disease of the skin.

Etiology *Molluscum contagiosum* is a contagious disease caused by a filtrable virus. Intracutaneous injections of a sterile filtrate of molluscum lesions suc-

cessfully reproduce molluscum lesions. Epidemics have been reported to occur in institutions, schools, bathing establishments, etc. It occurs equally in both sexes at all ages. Sweating, itching, and pediculosis favor dissemination of the

and comedones. Careful examination will readily identify these lesions.

Treatment The disease is treated by *evacuating the contents* of lesions by manual pressure and the application of *carbolic acid trichloroacetic acid or tincture of iodine* to the central cavity. Healing occurs within a week without scarring.

Where the lesions are numerous, disappearance of the lesions may result from the use of *green soap*. Vigorous *massage with sulfur ointment* will produce a similar exfoliation and disappear

ance of the lesions. Electrodesiccation is used but has no special advantage.

Local anesthesia (ethyl chloride) may be deemed necessary in children with a large number of lesions in order to cauterize these newgrowths.

The monopolar *high frequency current* on patients with numerous lesions has given excellent results in the hands of Goodman.

Sulfapyridine (2 to 4 gm [50 to 60 grains]) given over a period of two to three weeks, has been used successfully by Hill and Downing.

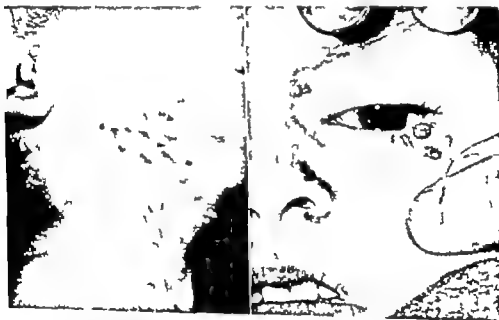


Fig. 353 *Monilium Contagiosum*. Note umbilication of lesions.

MONILIASIS

SYNONYMS *Oidiomycosis, pieb thrush, mycetozoa, saccharomycotic dermatitis, candidosis.*

The monilia are yeastlike fungi causing pathological lesions of the skin, mucous membrane, respiratory and gastrointestinal tract.

Varieties Monilia comprise a large number of organisms belonging to the

group of yeastlike fungi. There are probably more than thirty in the group. They grow readily at room temperature under aerobic conditions.

Incidence They are found on plants, animals, on the normal human skin and

lesions in affected individuals. The incubation period ranges from six weeks to six months.

Incidence. It occurs more frequently in children. It is more common in England than on the European continent or in America.



Fig. 351. Molluscum Contagiosum. Of thigh; single lesion; note umbilication.

Pathology. The lesion of molluscum contagiosum is a sessile lobulated epithelial tumor having a somewhat constricted neck. The growth has a fibrous capsule which sends thin septa between lobules. These septa are composed of keratin. The tumor consists of a group of enormously hypertrophied interpapillary processes of the prickle-cell layer which are known as molluscum bodies. The cells in the center of each process degenerate and stain in different colors as they get nearer the surface. The result of this degeneration is a yellowish

cheeselike disorganized mass known as the molluscum body. This mass of hypertrophied interpapillary processes raises up the surrounding corium and epidermis to form the outer layers of the tumor.

Symptoms. Molluscum contagiosum is characterized by a single papule or multiple rounded flat, pearly-gray cutaneous papules ranging in size from a pinhead to a pea. Each tumor has a central depression from which a cheeselike substance is expressed. The number of lesions varies as a rule from one to a dozen or more. Molluscum lesions occasionally occur in patches. The common sites of predilection are the eyelids, lips, arms, breasts, and genitalia and rarely on the scalp. The distribution may however be general. Lesions grow slowly until maturity is reached, when they remain unchanged for weeks or months. Newgrowths make their appearance at regular intervals, thus making it possible to see variously sized lesions at one time.

Whitfield reports a variety characterized by small, red acuminate papules having a translucent center which is visible only with a lens. Severe itching may be present. The sites of predilection of this variety are the back, shoulders, and chest.

Subjective symptoms may be absent in molluscum contagiosum except when the lesions are numerous and when they undergo inflammatory changes, when itching would be present.

Diagnosis. The diagnosis is recognized from the lesions which are characterized by globular rounded flat, pearly gray semitranslucent tumors, with a dark central depression. These lesions are pathognomonic and are unmistakable. The lesions resemble somewhat small fibromas, milia, verrucae,

and comedones. Careful examination will readily identify these lesions.

Treatment The disease is treated by evacuating the contents of lesions by manual pressure and the application of carbolic acid trichloroacetic acid or tincture of iodine to the central cavity. Healing occurs within a week without scarring.

Where the lesions are numerous, disappearance of the lesions may result from the use of green soap. Vigorous massage with sulfur ointment will produce a similar exfoliation and disappear

ance of the lesions. Electrodesiccation is used but has no special advantage.

Local anesthesia (ethyl chloride) may be deemed necessary in children with a large number of lesions in order to cauterize these newgrowths.

The monopolar high frequency current on patients with numerous lesions has given excellent results in the hands of Goodman.

Sulfapyridine (2 to 4 gm (30 to 60 grains)) given over a period of two to three weeks, has been used successfully by Hill and Downing.



Fig. 335 *Monilium Contagiosum*. Note stabilization of lesions.

MONILIASIS

SYNONYMS *Oidiotomycosis*, *spuma*, *thrush*, *mycetomycosis*, *saccharomycetic dermatitis*, *condilliosis*.

The monilia are yeastlike fungi causing pathological lesions of the skin, mucous membrane, respiratory and gastrointestinal tract.

Varieties Monilia comprise a large number of organisms belonging to the

group of yeastlike fungi. There are probably more than thirty in the group. They grow readily at room temperature under aerobic conditions.

Incidence They are found on plants, animals, on the normal human skin and

mucous membrane as well as on dead material. They are omnipresent.

Etiology. *Monilia albicans*, the most important species of the group, is generally accepted as the etiological agent of the various pathological conditions affecting the skin and mucous membrane.



Fig. 356: Moniliasis. Sharply margined reddish, scaly eruption.

However, it is seldom primary in nature, usually developing because the resistance of the tissue is diminished by other factors. *Monilia albicans* is more apt to be present in individuals whose skin is macerated as the result of frequent washing or prolonged immersion in water. Bartenders, waiters, washerwomen, and bakers are especially predisposed to this infection. Predisposing factors include obesity, diabetes, anemia, tuberculosis, cancer, and the chronic diseases. Patients with avitaminosis B are extremely vulnerable to this infection.

Pathology. The histopathology of moniliasis is similar to that observed in the various forms of dermatomycoses.

Clinical Forms. Lewis and Hopper list the following clinical forms of moniliasis:

Localized. Onychia and paronychia, intertrigo, erosio interdigitalis, perlèche, thrush, superficial glossitis, localized patches of eczema, water bed dermatitis, and vulvovaginitis.

Generalized. Widespread eruption usually associated with some of the localized forms. Monilids in which no fungi are found.

Systemic. This group includes cases of pulmonary and gastrointestinal infection. These may be associated with cutaneous infection.

Onychia and Paronychia

Paronychia is often the result of monilial infection and it is one of the most frequent diseases of the nails. It occurs at any age but most frequently among washerwomen or in those engaged in



Fig. 357: Moniliasis. Showing sharply margined, reddish patches, with scaly borders.

frequent immersion of the hands in water. It may occur from occupational injuries. Fruit-canners and cooks are frequently affected.

It is characterized by marked inflammation of the periungual tissue and is more marked in the lunula area and

gradually lessens toward the fingertips. Suppuration is often present between the nail and the nail wall. It is chronic and spreads from one finger to another. The nails may exfoliate. Pain is often severe. *Eroso interdigitalis* is frequently associated with this affection.

Paronychia is differentiated from trichophyton infection by the cushionlike thickening of the paronychia tissue, by the erosion of the lateral borders of the nails, by transverse ridges, and the gradual thickening and brownish discoloration of the nailplates.

Monilial onychomycosis is usually secondary to cutaneous or mucous membrane involvement. The differentiation between monilial and trichophytic onychomycosis can only be made by culture; they are clinically similar.

Intertrigo

This type of cutaneous moniliasis is the most common. The sites of predilection are the intergluteal, genitocrural, and submammary folds of the neck, and the interdigital areas. It is characterized by erythema, abrasions, and areas of maceration located on opposing surfaces. It starts either as a vesicle or erythematous macule which grows by peripheral extension forming erythematous, scaly, round, or polycyclic areas with well defined border. Hot weather, obesity, too tightly fitting garments are predisposing factors. If hyperhidrosis is present the skin is abraded and has a raw appearance with overhanging margins (Collarette). If the condition continues, papules, nodules, or necrotic ulcers may result.

In the female genitalia, the mucous membrane between the labia and about the clitoris is thickened and white. The cervix is often hyperemic and swollen and may be covered with

small vesicles or eroded. The vaginal discharge is thick and tenacious. The anus is usually grayish white, and flourishing may be present. In the male, balanoposthitis is usually present and a monilial urethritis is observed occasionally. Diabetic patients are most prone to this infection. Genital pruritus and genital dermatitis, in both male and female, are probably the result of a secondary monilial infection on the skin sprayed by voided saccharine urine. Vitamin deficiency and anemia are often associated. This type of moniliasis is also common in infants. It may be wide spread and is frequently associated with or preceded by mucosal thrush. The face and neck are often the only sites affected. Cleanliness, wearing of loose clothing, the use of a borated talcum dusting powder, and the avoidance of exercise in hot, moist weather are prophylactic measures.

Eroso Interdigitale

SYNONYMS *Monilia interdigitalis dermatitis*, *eroseo interdigitalis blastomycosis*.

This type of moniliasis is characterized by oval-shaped areas of macerated skin affecting the interdigital fold between the middle and ring finger. The eruption may spread to adjacent areas and affect other interdigital folds and nails. The lesions are inflammatory, sharply defined, superficial, and have undermined macerated borders beneath which monilial organisms are obtainable. This condition occasionally extends over the dorsum of the hand to the forearms. It occurs most often in laundresses and others whose skin is subject to maceration from water or strong alkalis. Similar types of eruption may involve the toes; if so, the fourth interspace is most often involved.

Clinically this condition is indistinguishable from dermatophytosis. Culture of the monilia is the diagnostic feature. Erosio interdigitalis is chronic and resistant to treatment. It becomes more amenable to therapy if the contributing etiologic factors noted above are eliminated.

may be on a nutritional or infectious basis. In the bacterial types, the causative organisms are monilia in some streptococci in others, a vitamin deficiency especially of riboflavin is causative at times. In elderly people it is due to malocclusion of dentures and salivary drooling while a sleep.



FIG. 358. Left: Erosio Interdigitalis Saccharomyces. Right: Monilia.

Perleche

SYNONYMS *Angula Infectiosa*
angular or commissa al cheilitis.

Perleche is an intertriginous inflammation of the labial commissures and is characterized by macerated, crusted and fissured lesions.

Varieties It occurs in an acute and a chronic form.

Incidence The acute variety affects children more often while the chronic form most frequently affects adults, especially women after the age of forty.

Etiology The etiologic factors can not always be determined. The epidemic and endemic cases seen in institutions

Symptoms The earliest lesions are ill-defined grayish white thickened areas with slight erythema occurring on the mucous membrane at the buccal commissure. These areas finally present a bluish white or mother-of-pearl color. An erythematous scaling dermatitis of the skin portion of the commissure associated with fissures, maceration and crust formation is usually present. The involvement is usually bilateral. In severe cases, deep wrinkles and transverse fissures causing discomfort and pain on masticating food are present. This disease affects chiefly children and being highly contagious, may spread to other

children in the same household or school. In adults, it usually occurs in cases of lowered resistance due to vitamin deficiency anemia, or diabetes, but also as the result of poorly fitted artificial dentures.

This condition must be distinguished from mucous patches and syphilitic

sized and shaped superficial adherent deposits resembling coagulated milk. These cream-colored or grayish-white plaques occur on the tongue, gingiva, cheeks, and pharynx. The plaques on removal reveal a red base. The fungus is readily demonstrated in the scrapings, both microscopically and culturally. The



Fig. 3-9 Perleche Complicating facial Impetigo.

papules. When secondary syphilis involves the mouth, generalized syphilids are almost always present. Interdigital ulcers and paronychia are often associated with perleche.

Monilliasis of the Mouth

Oral lesions caused by monilia include thrush, glossitis, and stomatitis.

Thrush is characterized by variously

circumoral skin may be involved and is characterized by the presence of furfuraceous scaling (pityriasis alba). Marasmic infants if infected with thrush are also often affected with cutaneous lesions, especially in the groin, the diaper area, axillae, and a generalized eruption is occasionally present. The latter is characterized by small white pustules which on drying form macerated, scal-

ing patches. The disease affects infants and occasionally adults. It is not an uncommon affection in pellagrins. Epidemics occur, the organism being carried by unclean utensils, or by direct contact (kissing).

Moniliasis of the oral cavity is similar to that seen in children, however it is often chronic and is seen more often on



Fig. 360: Perleche (bilateral). Also known as angular cheilitis, due to malocclusion from ill fitting dentures.

the tongue and gums. Monilial glossitis occurs in pellagra, sprue, and patients suffering from avitaminosis. The acute symptoms include hyperemia of the fungiform papillae and aphthous ulcers. The chronic cases are characterized by atrophy of the papillae and a smooth tongue. Pain, burning sensation, extreme sensitiveness to hot fluids, spices, and tobacco are the principal subjective symptoms.

Moniliasis of the mouth is frequently associated with perleche. Associated lesions also include those involving the perianal region and eczematous areas on the hands and forearms.

Water-Bed Dermatitis

Intertriginous eruptions of the groins and axillae are common in those kept on water beds or in continuous baths. In intertriginous eruptions may also occur on the palms and soles.

Monilial Pruritus Ani et Vulvae

Monilial infection is a common cause of pruritus ani and pruritus vulvae. Monilial infection of the vagina may be asymptomatic; it occurs in infants, virgins, and women of all ages. In symptomatic types the first symptoms are itching, burning, and smarting of the lower part of the vagina and vulva. This infection may continue over the years or it may spontaneously disappear. Menstruation, as well as the delivery of pregnancy, has a beneficial effect on this condition.

Sutton and Sutton describe the following types:

- 1 Creamy vaginitis resembling oral thrush with painful and reddened mucosa.
- 2 Creamy vulvitis, with considerable discharge, not much redness, with or without intertrigo which consists of small grouped shallow vasopapules.
- 3 Ulcerative vulvitis, severe superficially ulcerated with pain, lymphangitis, and vaginal adenitis, possibly with mycotic infection of the bladder.
- 4 Pseudoleukoplakic vulvitis, in which the mucosa is whitish and opaque and the crusts are hard to remove, but no wrinkling papillomas, or hyperkeratosis exists.
- 5 Eczematoid vulvitis, with a vesiculopustular eruption consisting of small punctiform erosions, spreading and resembling intertrigo.
- 6 Mycotic pruritus of the vulva with few erosions, and little discharge.

7 Vesiculopustular cutaneous form, manifesting disseminated involvement of external integument with groups of small vesicopustules.

8. Cutaneous intertriginous eczematous form, intertrigo-like, with or without demonstrable organisms in the genito-crural plica, the inflammation spreading to the yuba, anal region, and medial surface of the thighs, with but little involvement of the vulva.

9 Inconspicuous vaginitis with disseminated cutaneous involvement in the form of patches of intertriginous dermatitis with small vesicopustules.

10 Monilids, often consisting of pompholyx-like patches of thin deep-seated vesicles which may absorb and result in scaling or which may result in oozing eczematous dermatitis.

Monillial Infantile Eczema and Dyshidrotic Monilliasis

Infantile Eczema This is characterized by deep-red, oval, circumscribed, and coalescing patches of oozing dermatitis, which is usually caused by monillial infections and probably originates in the birth canal.

Dyshidrotic Monilliasis This form of monilliasis is rare. It may be the cause of pompholyx-like lesions.

Systemic and Generalized Monilliasis

This condition rarely occurs and small children are more liable to infection than adults. In children it usually follows thrush where absorption from these areas to the blood stream is probably the means of diffusion. The symptoms of systemic involvement resemble a low grade septicemia. The condition is usually of brief duration. In children, toxemia and fatal alopecia may follow cutaneous and

mucous membrane involvement. The majority of cases end fatally. On autopsy the following organs usually show the presence of monillial foci: pharynx, esophagus, kidney, liver, spleen, brain, meninges, and rarely the lungs. If adults are affected, bronchopulmonary symptoms are often present and the condition simulates tuberculosis of the lungs. The condition invariably ends fatally.

Monilids

SYNONYMS *Levureids, monillid dermatomycosis.*

Monilids are the cutaneous toxic manifestations, on an allergic basis, produced by a monillial infection and disseminated by the lymph and blood stream. The initial lesions are small, closely aggregated intraepidermal vesicles occurring on the hands, legs, flexor surface, and those areas common to monillial infections. They are clinically and histologically similar to monilliasis and differ only in the absence of the monillial fungus. In some cases the affections resemble that of seborrheic eczema. The scalp may be covered with greasy crusts and the mouth and nose may be the site of erythematous areas with crusted borders.

Foci of monillial infection in the vagina, tonsils, mouth, nails, auditory canal, the glabrous skin, and the gastrointestinal tract may be the sites for dissemination of the allergens producing monilids.

Monilliasis in General

Diagnosis In cases of doubt in diagnosis, microscopic examination of the epithelial threads and scrapings after treatment with potassium hydroxide will disclose the etiological organism. The organisms are gram-positive and are readily stained by any of the aniline dyes. The organisms appear as long, delicate, mycelial threads, usually branched, and

MYCOSIS FUNGOIDES

SYNONYMS: *C annuloma fungoides*, *fibroma fungoides*, *lymphodermis perniciosa*, *granuloma sarcomatodes*

Mycosis fungoides is a chronic inflammatory disease of the skin characterized by areas of erythema infiltration tumor formation and ulceration

Etiology The cause is unknown



Fig 361 Mycosis Fungoides. "Tumor d'emblee" type with central slough and resultant ulceration.

Symptomatology The disease develops slowly and is marked by three well defined phases: the stage of erythema (pre-fungoid), the stage of infiltration, and the fungoid or tumor stage. The three phases do not always occur in this sequence. Occasionally the tumor stage may be the only manifestation of the disease. This type is referred to as "mycosis d'emblee".

The premycotic stage is characterized by circumscribed plaques of dermatitis

usually accompanied by mild to severe itching. Itching is, in exceptional cases, absent. It may in early cases, be the only symptom. The patches resemble eczema, psoriasis, parapsoriasis, or urticaria. This stage may continue for months or years before signs of infiltration develop.

The stage of infiltration is characterized by slightly elevated plaques which are bluish red. These areas may remain discrete or coalesce and form circinate



Fig 362 Mycosis Fungoides. "Tumor d'emblee" type with single growth on forehead; ulceration and additional lesions developed later; characteristic histology.

lesions. These stages may persist for years.

The fungoid or tumor stage is characterized by the development of nodules or tumors on the areas of inflammation, but may develop on normal skin. The

tumors are usually dusky red, and the surface often becomes necrotic. They vary in size from that of a bean to that of an orange; they may be sessile or pedunculated. Tumors may be numerous and may appear anywhere on the skin surface and occasionally on the

three stages may regress. New lesions replace old ones at previously involved areas. The lymph nodes are only occasionally enlarged. Ulceration is the usual termination.

Diagnosis The intense pruritus in early stages of mycosis fungoides is



Fig. 363 Mycosis Fungoides.

tongue. Partial or complete alopecia often occurs on the scalp as well as elsewhere. Pruritus is usually absent in this stage of the disease. This tumor stage may develop as early as a few months after the stage of infiltration.

The cutaneous manifestations of these

characteristic of the disease. The tumor stage is differentiated from nodular leprosy and multiple sarcoma. Biopsy is necessary to establish a diagnosis.

Pathology The histopathology consists of a polymorphous cellular infiltration within the lymphatic spaces be-

tween the connective-tissue bundles and around the blood vessels. Microabscesses filled with lymphocytes are characteristic.

The structure in the tumor stage closely resembles a sarcoma or an infectious granuloma.

Acanthosis, intercellular edema and parakeratosis are present in the preinfiltrative stage.



Fig. 364: Mycosis Fungoides.
(Courtesy of Dr. Ralph Bernstein.)

Prognosis This disease usually ends in death. The course of the disease is about five years; however, some cases have lasted forty years.

Treatment Roentgen ray therapy is palliative, produces regression of lesions, and controls itching. The dosage depends entirely on how sensitive the lesions are; the cases must be individu-



Fig. 365: Mycosis Fungoides.
Note tumor formation.

alized since some respond quickly to small and others only to large doses. Maximum results should be obtained with minimum radiation.

Inoculation with malaria and other forms of artificial fever therapy have been successful in some cases. The injection of *chaulmoogra-oil esters* over a long period of time of *Coley's fluid*, *benzene* and *arsenic* have been favorably reported. According to O'Leary *chaulmoestrol* or *ethyl chaulmoograte* is best given (1 cc. ascending to 6 cc. intramuscularly daily) for thirty injections during the course of fever therapy. It may be given again to those who have become unresponsive to x-ray therapy. Local therapy is valueless and is limited to antipruritic medication.



Fig. 346 Myxoid Erythema. (Courtesy of Dr. Carroll S. Wright.)

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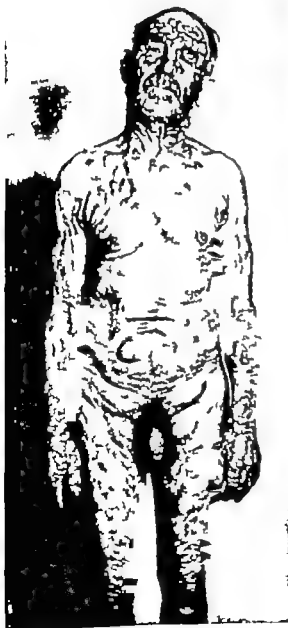


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Local therapy is valueless and is limited to antipruritic medication.

MYXEDEMA

SYNONYMS *Cachexia thyroidea*, *cachexia strumipetec*, *cretinoid edema*, *Gull disease*.

This is a constitutional dystrophy characterized in part by a more or less generalized edematous swelling, and later by thickening with induration of the skin and subcutaneous tissues.

Etiology The disease is not common. It is seen at any age, but especially in the female adult. Heredity is a factor in some cases. The disease follows atrophy or surgical ablation of the thyroid gland.

Pathology The histologic cutaneous features are essentially those of dermic edema due to paramucin, obliterative end arteritis, and later to an increase in collagenous tissue and subcutaneous fat.

Symptoms The subjective symptoms are chiefly sensations of cold. In well-marked instances, the skin is swollen, waxy white, dry shiny and sometimes scaly. The edema is firm and does not pit on pressure. There is a lack or loss of sweat and sebaceous secretions. The hair becomes dry and sparse, and there is a tendency to alopecia, especially of the scalp and outer borders of the eyebrows. The nose, cheeks, lips, and the face as a whole become swollen, as do the neck and extremities.

In simple hypothyroidism, seen at puberty and the menopause, the foregoing symptoms vary with the degree to which the thyroid fails to maintain normal metabolism.

The extracutaneous disturbances consist in mental hebetude, slowness of speech, heart, and physical activities, and a decrease in body temperature and the basal metabolic rate.

Circumscribed Myxedema (Localized Myxedema) The relationship of this form to hypothyroidism is not clear.

Nodular and papular lesions are observed here and there on the body in the presence or absence of myxedema, especially in hyperthyroidism (Graves disease) and following subtotal thyroidectomy for exophthalmic goiter. In some,



Fig. 367 Circumscribed Myxedema. The leg is a common location; the non-inflammatory localized, firm, non-pitting lesions are usually not so sharply levated. (Courtesy of Dr Hans J. Schwartz.)

Neumann, under the term "Lichen myx edematosus," has reported lichenoid lesions described as small, round, waxy to red translucent, discrete, and coalesced. In others, one finds discrete or diffused and elastic swellings on the scalp, face, upper extremities, scrotum, and but

NAEVI

The term "naevus" is generally used to designate all circumscribed cutaneous malformations of embryonic genesis. The term "mole" is used for both cellular and purely pigmented naevi. Generally speaking, naevi are so common that it is rare to find a person with out at least one of them. The tendency to them appears to be hereditary. In some families the same mark may appear in successive generations, but a child may be born with a totally different type of lesion. A person may have many different types. Patients with multiple naevi often show other congenital malformations, such as developmental arrests and hydrocephalus. Naevi may be present at birth or develop shortly or many years afterward. They remain stationary, enlarge or even disappear partly or completely. They appear in the form of persistent spots or as real neoplasms. The term "nevic dermatosis" has been applied by Darier to those cases in which instead of isolated lesions here and there, one finds numerous lesions of different types with or without other malformations. Typical examples of nevic dermatoses are neurofibromatosis of Von Recklinghausen (p. 827) and xeroderma pigmentosum (p. 843).

Classification. For clinical purposes, it is usual to classify naevi into two main groups (Brocq)

I. Nonvascular

A. Naevus pigmentosus

B. Naevus tuberosus

1. Soft tuberosus naevus

2. Moles of naevi

(a) Moles of pendulum

(b) Moles of fibrous

(c) Moles of fibrous granular

2. Hard tuberosus naevus

A. Adenomatous naevi

(a) Sebaceous adenoma

(b) Hidradenoma

(c) Epithelioma adenoide cysticum

II. Vascular

A. Hemangioma

B. Lymphangioma

Nonvascular Naevi

Naevus Pigmentosus

SYNONYMS *Naevus spilus, beauty spots, lentigo, liver spots.*

Pure pigmented naevi range from brownish to black, and are of various sizes and shapes (irregular oval, and round). These naevi are macular with a smooth surface and no thickening of the skin, although palpation often suggests slight elevation. They are commonly multiple and scattered over the skin. They are the smooth flat naevi of a slate-blue to blue-black color which are usually the precursor lesions of melanoma. Melanoblasts are found in pigmented naevi, in varying amounts, in both epidermis and dermis. According to Traub dermoepidermal or the so-called "junction naevi" are superficial, pigmented lesions in which collections of naevic cells are limited to the upper portions of the cutis or occur as intraepidermal nests with occasional downgrowth into the cutis. They are benign but potentially malignant. They are smooth light brown to brownish black, and usually devoid of coarse hair.

Blue Naevi. Blue papules or nodules (blue naevi of Jadassohn) sometimes occur—usually on the face—and are essentially benign but often confused with melanocarcinoma. Histological examina-

tocks these swellings are nodular and vary in size and color from skin toned to reddish. In a third commoner type, irregularly defined swellings limited to the shin areas are observed these thickened areas are brownish or yellowish and noninflammatory and vary in size. The histologic findings are similar to those found in myxedema.

Diagnosis In well marked cases of myxedema diagnosis is not difficult. In *acromegaly* the bones are greatly enlarged in *elephantiasis* the swelling is regional and the basal metabolic rate is unaffected. In mild cases, in patients who tire easily or are sensitive to cold weather and in those with but dryness and roughness of the skin, dryness of hair and its sparseness in the axillae, pubes and eyebrows as well as the splitting and brittleness of the nails, a study of the blood cholesterol of the basal

metabolic rate, and a therapeutic test with thyroid extract are necessary for the diagnosis. The diagnosis in circumscribed myxedema depends upon the history, physical examination and a histologic study.

Prognosis Unless treated properly the disease in complete thyroid atrophy or ablation is chronic and progressive. Partial thyroid secretory deficiency may or may not be persistent.

Treatment *Thyroid gland extract* in adequate later stabilizing doses, is specific in generalized myxedema. The drug should be given to control symptoms rather than to control the basal metabolic rate level. The daily dose is usually 0.1 gm ($1\frac{1}{2}$ grains). This dose is increased within three to four days until symptoms disappear (about two months) and then it is reduced to a minimum daily maintenance dose.



Fig. 369 *Left* Hairy Naevus and Congenital Angioma. Lower thoracic region.
Right Hairy Naevus.



Fig. 370 Mongolian Blue Naevus. (Courtesy of Dr. Carroll S. Wright.)

tion reveals the pigment deposition deep in the cutis. This type must not be mistaken for the *Mongolian blue* or *pigmented spot* that is occasionally observed at birth in infants of the darker races and in Japanese and Mongolian infants in particular. In these latter bluish or slate-colored areas are not infrequently seen in the skin of the lumbosacral re-

gion the buttocks, and the shoulders. They generally disappear at about the sixth year of life. In this condition, an unusually large number of normal melanoblasts are found in the derm. The dopa reaction is positive both in blue naevi and in Mongolian spots.

zosteriform one (*spilus zosteriformis*). These types often appear some years after birth and even in the adult, and often after exposure to sunlight or x rays. Pigmented linear naevi may change spontaneously in size and color or under the influence of light and such physiological processes as pregnancy and the menopause.



Fig. 368 Naevi.

Spilus Linearis *Spilus Zosteriformis*. In some instances, naevi have a linear arrangement (*spilus linearis*) or a

Incontinentia pigmenti *Incontinentia pigmenti* (Bloch-Sulzberger) or chromatophore naevus (Naegeli) is a rare nevroid pigmentary anomaly which appears during the first years of life in the form of sharply outlined irregularly shaped and irregularly distributed areas of brownish pigmentation on the trunk or extremities. Histologically coarse granules of pigment are found largely

Ephelides are small, usually numerous, and have a surface that is smooth, non scaly and nonelevated. They are yellowish or brownish, and round, oval, or irregularly shaped. They generally become more marked in the spring and summer under the influence of sunlight.



Fig. 373 Melanoma. Patient had pigmented mole for years; growth developed after constant picking at it.

If they persist during the winter they may be considered to be of the nature of lentigines. They are usually located on all parts of the face or the dorsum of hand or forearm, and on the shoulders. On histological examination, the pigment in ephelides is found to be chiefly in the prickle-cell layer of the epidermis. Although freckles and lentigines are ordinarily easy to differentiate it must be admitted that there are transitional forms. Lentigo maligna is a malignant melanoma. For their treatment, see Ephelides, p. 259.

Liver Spots. Liver spots are round, oval, or irregular and are brownish or

liver-colored. They vary in size, and are occasionally as large as the palm of one's hand. They are usually seen on the trunk and are really large pigmented naevi. They are in no way different from those commonly seen in the abortive forms of neurofibromatosis, except that ordinary liver spots occur singly or doubly (there may be several) in the absence of the other cutaneous disturbances characteristic of neurofibromatosis. They are not indicative of liver disease. They apparently differ in no way from lentigines except in their size and color (See Chloasma p. 219).

Treatment. It is not necessary to remove all pigmented naevi, but those apt to undergo constant irritation should



Fig. 374 Melanotic Whitlow Of great toe

be removed. Insufficient removal under these circumstances, or under those in which cosmetic considerations are permitted to be paramount is dangerous, and may lead to malignant changes, commencing in the remaining area. In

in the phagocytic chromatophores and not in the pigment forming melanoblasts. Nevic cells are not found. The dopa reaction is negative in the cutis.

Lentigo. These are frecklelike, brownish naevi appearing sometimes after



Fig. 371: Lentigo. Followed a severe sunburn six years previously.

birth and often under the influence of sunlight. They are permanent and usually show intradermal naevoid cells. They are often confused with the ordinary lighter-colored freckles or ephelides. Lentigines are apt to develop on any part of the body and on any type of skin. They vary in size and number, are more apparent in winter, are sharply defined and flat or slightly elevated.

The pigment on histologic examination, extends to the connective tissue; it is pigment producing (Dopa positive reaction). The lesions are apt to be permanent and malignant degeneration has been observed. They may develop suddenly after a summer's tanning of the skin, a febrile bout or in association with such conditions as Addison's dis-



Fig. 372: Lentigo. Developed after severe sunburn; patient showed no lentigines or ephelides on the face.

ease and neurofibromatosis. Ephelides are not ordinarily considered naevi, but are discussed here owing to their constant hereditary and familial tendencies.

used early. It may be used prophylactically. The dosage is three-quarters erythema unit, well screened, at three-week intervals for from four to six exposures.

Naevus Tuberosus

SOFT VERRUCOSE NAEVI

Description: These naevi are warty and are produced by alterations in other than the pigmentary elements of the skin. In some, hair occurs in a varying amount. In others, there is both hair and pigment (*naevus pigmentosus et verrucosus et pilosus*). In *naevus lipomatous*, there is an excess of fat and connective tissue. The growth may become very large, with a smooth, yellowish white surface and the nonfluctuating consistency of the ordinary lipoma.

In the infant, as a rule, soft verrucose naevi are flat, and show only slight infiltration. As the child ages, they increase in size and thickness, and the covering skin becomes marked by numerous small and irregular elevations.

The pigmentation varies with each case; ordinarily it is pink or bluish red. The lesions vary in size from a pinhead to a large pea or larger and occasionally spread out in the form of a plaque; they are often covered with hair. Sometimes, especially after the age of forty, they undergo malignant degeneration.

The first sign of malignant degeneration may be persistent itching. Later there are swelling of a part of the lesion, induration of its base, and irregular ulceration, which bleeds easily and is covered with an adherent blackish crust. Whether it ulcerates or not, the lesion rapidly increases in size. The surrounding lymphatics often show the first signs of metastasis, with involvement of the draining glands. *Naevocarcinomas* are almost always heavily pigmented.

Treatment. *Surgical excision* is the method of choice. These naevi may also be destroyed by *electrocoagulation* or by *negative electrolysis*. In the case of a hairy naevus, the hairs should be removed first by electrolysis. It often happens that, as each hair is thus removed, the naevus shrinks until, with the final hairs out, it becomes merely a flattened, hairless crater.



Fig. 377. *Molluscum Fibrosum.*

When the naevus is rather large and electrolysis is to be performed, the needle attached to the negative pole is thrust through the base of the naevus, and a current of from 2 to 3 milliamperes is applied. As one feels the needle loosen, following liquefaction of the region surrounding it, the current is turned off, the needle is withdrawn, and is thrust again through the base of the naevus 4 or 5 mm. from the site of the first penetration. The smaller the lesion, the less often is it necessary to do this. For larger lesions, the procedure may require repetition in from ten to fourteen days.

this regard blue-black moles especially when located on the feet are particularly dangerous and apt to eventuate in malignant melanomas. Pigmented moles may be removed by one of several methods by solid carbon dioxide (applica-



Fig. 375 Melanoma. Begun a pigmented naevus on center of abdomen. Others are metastatic.

tion of a carefully shaped pencil for from three to five seconds) by galvanocautery by electrodesiccation or coagulation by negative electrolysis or by surgical extirpation. Wise and Fox state that solid carbon dioxide gives the best results in pigmented naevi. Hair if present, should be shaved off before applying the snow and the entire lesion treated at one sitting if possible. Caustics are not advisable except when the lesion is very superficial, when the application of pure phenol or trichloroacetic acid is often sufficient. One of these is applied directly to the lesion with a pointed applicator

until the naevus becomes white. For large lesions, and especially those that show perceptible elevation on palpation, surgical excision is the method of choice. A mole which changes in size or pigmentation or has commenced to grow spontaneously as a result of injury or injudicious treatment, requires early radical excision. (See Melanoma, p 203.)

Keloids may follow the removal of naevi, and do not disappear spontaneously. Roentgen ray therapy ordinarily causes them to vanish and should be

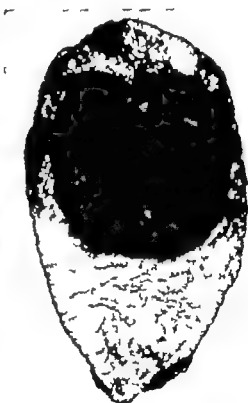


Fig. 376: Melanoma (after excision). Eight months after piecemeal removal of a pigmented naevus of forearm ("physician used an 'electric needle' three times weekly for four months, at the end of which time there was some pigment still remaining"). The lesion began to enlarge peripherally and to thicken in the center; black pigment reappeared in all areas of the scar; entire lesion was widely excised; axillary lymph nodes became palpable six months later; death from cerebral metastasis.

some cases, an infectious rather than a naevoid origin is suggested. Occasionally they develop during the later months of pregnancy (molluscum gravidarum) disappearing partly or completely after parturition. The lesions often fall off spontaneously but they may be easily destroyed by means of the

Molluscum Fibrosum Generalisatum (Neuroglomatosis; Neurofibromatosis of Von Recklinghausen) There is an abortive form of this condition in which no molluscoid lesions are present. In the abortive form, the lesions are café-au-lait pigmented spots, either irregularly rounded or oval, smooth and flat. There



Fig. 381 Von Recklinghausen Disease Note pigmented areas which, in some cases, are the only signs of this affection. (Courtesy of Dr Jacques P. Goquerel.)

galvanocautery or by use of electrodermication

Molluscum Fibrosum One, two, or more of these growths may occur. They are rather large painless, pedunculated, and soft and usually appear on axillae, neck, chest, thighs, and labia majora. They may be flat and ridged, like a dried ramus, or as large and round as peas or larger. They feel and are soft and pasty

may be several large ones or numerous small ones, generally on the trunk. However in the typical case, firm nodular growths, associated with such pigmented spots, occur along the course of nerves, and are, in addition, associated with numerous flaccid or distended skin tumors. The cutaneous growths (perineuromas) vary in size and shape, and are soft, painless, pedunculated or sessile and either barely perceptible or

The growth swells after treatment, but shrinks later. The treated lesion should be kept dry and aseptized with powdered *thymol iodide* or powdered *sulfathiazole*.



Fig. 378: Neurofibromatosis. Note "café-au-lait" spots.

DERMATOSIS PAPULOSA NIGRA (Castellani)

This is a common disorder in both male and female negroes. The lesions are pinhead or larger sized round discrete, skin-colored or hyperpigmented single or multiple papules. They are commonly located on the cheeks below the eyes and on the malar areas. They are benign.

MOLLUSCOID NAEVI

Molluscum Pendulum Growths of this type are very small and usually

numerous. About the size of a pinhead, they are pedunculated and painless, and occur on various parts of the body. They are common on the neck and upper chest of women past forty years or after the menopause. They often occur in several women of the same family. In



Fig. 379: Neurofibromatosis. Note "café-au-lait" spots: few or many of these spots and no tumors constitute the abortive form of this disease.



Fig. 380: Neurofibromatosis (Von Recklinghausen) Abortive form. The lesions consist of numerous, arrowed sized, pigmented, "café-au-lait" spots only. The patient was dull mentally and poor in her school work.



Fig. 381 Neurofibromatosis (Von Becklinghausen) (Courtesy of Dr. Carroll S. Wright.)

as large as a hen's egg or larger. Hundreds are usually present on the trunk and extremities. The covering skin is thin but of normal color. Sometimes on pressure, the tumors can be pushed

Treatment. In some instances, the larger and even the smaller lesions must be and can be removed by *surgery* or by *electrocoagulation*. No other therapy is known.

HARD VERRUCOSE NAEVI

SYNONYMS: *Horny naevi, circumscribed congenital hyperkeratosis.*

Description. These naevi are flattened elevations, varying in size. Their color may be either grayish black or



Fig. 382. Linear Verrucous Nevus.

down into the derm as through a hermal orifice. They occasionally undergo sarcomatous degeneration primarily or secondary to incomplete removal. Physical and mental disturbances are the rule in neurofibromatosis: poor memory, speech defects, and decreased intelligence



Fig. 383. Epidermodyplasia Verruciformis. Lesions on dorsa of hands in a man born of a consanguineous marriage whose sister had the same condition. The lesions were generalized and appeared first at the age of nine years. The patient had squamous-cell epithelioma over the left parotid area. (Courtesy of Dr. Oswaldo G. Costa.)

that of the normal skin. The elevations are covered by hyperkeratotic epidermis. Sometimes they are arranged in a linear or zosteriform fashion and unilaterally (naevus verrucosus unius lateralis). In the linear forms, the elevations may be flattened in such a man-

EPIDERMODYPLASIA VERRUCIFORMIS (Lewandowsky-Lutz)

A rare dermatosis usually appearing shortly after birth or before puberty characterized by dull pink to brownish numerous, flat topped, warty lesions, closely resembling flat juvenile warts.



Fig. 385 Epidermodyplasia Verruciformis. Lesions on lower extremities.
(Courtesy of Dr. Oswaldo G. Costa.)

ner that they will resemble the papules of lichen planus. They may occasionally form keratodermic bands over the forearms, palms, and palmar surfaces of the fingers.

Treatment This consists in destroying the lesions with the *electrocautery* or by *electrocoagulation*.

The lesions are distributed, usually symmetrically and most profusely on the face, neck, and extremities, especially the dorsa of hands and feet. By confluence a number of them may form lichenified patches. Epitheliomas, developing at site of preexisting lesions on the forehead and temple, have been ob-

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Fig. 345 Epidermodyplasia Verruciformis. Lesions on lower extremities.
(Courtesy of Dr. Osvaldo L. Costa.)

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Treatment This consists in destroying the lesions with the electrocautery or by electrocoagulation.

The lesions are distributed, usually symmetrically and most profusely on the face, neck, and extremities, especially the dorsa of hands and feet. By coalescence a number of them may form lichenified patches. Epitheliomas, developing at site of preexisting lesions on the forehead and temple, have been ob-



Fig. 386 *Left: Nevus Vasculosus. Right: Nevus Hemangioma.*



Fig. 387 *Hemangioma. Right Of gums.*

acred The histologic changes, according to Wise and Satenstein, consist of acanthosis, disintegration of cells and nuclei, and the presence of clear or vacuolized cells in the basal epidermic layer. The condition must be differentiated from juvenile flat warts and from actrokeratosis verruciformis (Hofl). Histologic study may be necessary although in Hofl dermatosis, lesions are found on the palms and soles and none on the face. Treatment is of little avail.

ADENOMATOUS NAEVI

The term adenoma is generally used to designate benign epithelial new growths

originating in and having the structure of the cutaneous gland from which they grow. It should be noted that all appear to originate in an embryonic nest.

Varieties There are the following: sebaceous adenoma (p. 67) hidradenoma (p. 400) and epithelioma adenoides cysticum (p. 335).

Vascular Naevi

Types There are two major types of vascular naevi, but the elements of both may be present in a given lesion.

1. Blood vascular (angioms or hemangioma)
 - (a) *Kavernous angioma*
 - (b) *Hereditary telangiectasia*



Fig. 358. Hemangioma. A. Male, age one year. Hemangioma approximately 1.5 cm. in size located in center of upper lip. Destroyed by electrodesiccation under procaine anesthesia. B. Result four months later. C. Twenty years later. (Courtesy of Dr. George E. Piller.)

- (c) *Naevus flammeus*
- (d) *Naevus tuberosus vascularis* (angoma tuberosa)
- (e) *Naevus cavernosus* (angoma cavernosum)
- 2 Lymph vascular (lymphangioma)

Hemangioma

The pure hemangioma is a congenital newgrowth due to hypertrophy and dilatation of the cutaneous (sometimes the subcutaneous) blood vessels. It is a localized lesion and varies from purplish to red.

NAEVUS ARANEUS (SPIDER NAEVUS)

Description Characteristically seen on the face this lesion has a central portion about the size of a pinhead that is made up of a slightly elevated and dilated blood vessel from which radiate several or more variously sized telangiectases.

Treatment This naevus is successfully treated by electrolysis. The electric needle attached to the negative pole is introduced superficially into the central spot and a current of from 1 to 2 milliamperes is applied for from five to twenty seconds. Occasionally this treatment requires repetition in three or four weeks. At times, fulguration may be used with success.

HEREDITARY MULTIPLE TELANGIECTASIA

SYNONYMS *Recurring epistaxis with multiple telangiectasia of the skin and mucous membrane* (Osler) *Rendu Osler Weber syndrome, hereditary hemorrhagic angiomatosis, familial or hereditary hemorrhagic telangiectasia.*

Symptoms This syndrome is a congenital familial hereditary affection due to a malformation of the capillaries and their surrounding supporting tissues. Males and females are both affected, and apparently either parent may transmit

the disease. The telangiectases are probably the result of a congenital defect of development of the terminal capillary loops.

Recurrent bleeding generally from the nose, is one of the earliest signs of this

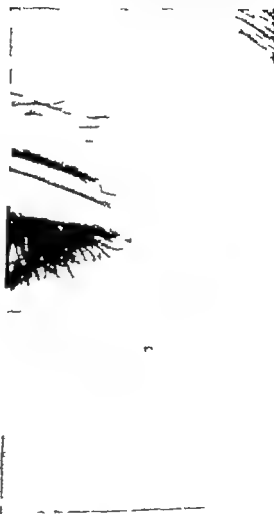


FIG. 389: Spider Naevus (naevus araneus) (Courtesy of Dr. Jacques P. Guequiere.)

affection. Later a variable number of telangiectases, stellate angiomas, ruby points, and angiomas which bleed easily develop especially on face and tongue. Abortive cases with but few such lesions are not rare. Bleeding from the telangiectases, especially those in the mouth, nose, and gastrointestinal tract, may lead to serious anemia.

Treatment The chronic anemia in this condition can be controlled by oral administration of iron. Kushan obtained an excellent result with *rustin* (40 mg three times daily)



Fig. 390 Hereditary Angioma-tosis. Multiple pigmented and larger-sized angiomas of tongue.



Fig. 391 Naevus Flammeus. With small aberrant element of purplish hue. Radium is of use in these types.

NAEVUS FLAMMEUS (PORT WINE STAIN)

Description This type is common on the nape of the neck of the newborn, and in this location often disappears spontaneously with age. It is less common elsewhere, but when seen is usually on the face or neck. It varies in size from that of a pea to involvement of



Fig. 392 Naevus Flammeus. A At birth. B Same lesion showing spontaneous development to cavernous and tuberosity-type angioma six months later. Markedly improved by surface radium with radium plaques and with intratumorous injections of quinone and urethane.

an extensive area. They are dark to bright red, smooth flattened and level with the surrounding skin. They are sometimes slightly elevated over their entire surface or only here and there. The elevations, when present, are soft nodular or warty projections. These flat vascular naevi partly disappear under pressure. According to Virchow these naevi are due to dilated dermic veins, whereas the arteries and capillary net



Fig. 393 Angioma.



Fig. 395: Tuberosus Angioma.
Of scrotal in



Fig. 391: Tuberosus Angioma.



Fig. 396 Angioma.

work are almost normal. They develop shortly after birth, may enlarge as the months pass, and may change into the tuberous type; by the second year of life, they have reached full development. They may then persist as they are or even gradually pale and disappear entirely. *Vaccus anemicus* (Vörner)

hours; it may be repeated in six to twelve weeks. *Radium* will benefit those which show the slightest elevation but neither it nor the x rays should be used in the pure flat types. Port wine stains can be readily concealed with suitably tinted *pastes* or *creams* and the commercial preparation *Cover-mark* Tat



Fig. 397 Tuberous Angioma. Left Extensive lesion of face before ulceration due to local infection. Right Showing curative effects of local infection, eight months later.

usually associated with port wine stains, is a well-defined white spot in which there is a genetic lack of vessels.

Treatment Small lesions can be removed by means of electrolysis and fulguration. Therapy in extensive lesions often gives unsatisfactory and poor results. However blistering doses with the cold quartz lamp will often cause some fading of the color. Good results with thorium x varnish (alpha rays, 1000 to 2000 electrostatic units) have been reported in some cases. It is painted on and removed in twenty four

hours; it has also been used for the purpose in some cases.

NAEVUS TUBEROSUS (ANGIOMA TUBEROSA, STRAWBERRY MARK)

Description These usually appear shortly after birth. Often insignificant at first, they frequently grow rapidly within a few weeks. These angiomas form well-marked elevations above the level of the surrounding skin. The growth varies in size and extent. Their color is bright red to bluish red. They are smooth or irregular and often en-

large temporarily on exertion struggling or crying. Tuberous angiomas can be reduced by pressure. Various degrees of a cavernous angioma may complicate the lesion. Infected at times, they then tend to ulcerate and undergo



Fig. 398: Cavernous Angioma of tip of nose. Lymphangiohemangioma on dorsum of tongue.

spontaneous cure with scar formation. In other instances they reach a certain size and then remain stationary. At times they pale and finally disappear completely.

Senile angiomas or ruby points (Darier) are pinhead or slightly larger angiomas which appear on the body and extremities of persons past the age of forty. They are considered late vascular naevi.

Treatment. The type of therapy selected in this and in cavernous angioma will vary with the age of the patient and with the location, size and extent of the lesion. It will also vary with the lesional evolution (studied over a period of three months) and the patient's

willingness to cooperate. It is well to remember that small, slow-growing lesions in infants often regress spontaneously. Certainly such lesions are rarely seen in school children or adults. *Lester Anderson* and others believe that tuberous angiomas and many cavernous angiomas involute spontaneously. Even though they may grow rapidly for a few months they cease enlarging by the end of six to twelve months, according to these authors. *Pfähler* states, and I agree with him, that one should not wait for spontaneous cures. However in a given case, the following procedures may be



Fig. 399: Cavernous Angioma. Age four years. Cavernous and tuberous portions of angioma obliterated with sclerosing injections and surface radiation with radium; plastic repair of cleft to be attempted at age of eight or nine years.

indicated because they are effective and conservative. For small circumscribed lesions, constant application over a long period of time of flexible collodion is occasionally successful. The collodion

should be applied every twenty-four to forty-eight hours. The fine galvanocautery electrofulguration, and negative electrolysis under local anesthesia also may be used for small lesions. The carbon dioxide pencil is of value in practically all tuberous naevi, especially for those on the scalp and eyelids. The pen

is usually sufficient to cure the naevus.

Sclerosing injections have been used recently with a considerable degree of success. However they are more valuable in cavernous types. Andrews advises guaiac and urea hydrochloride and in my hands it has proved of great value. A 1 per cent solution is used, at

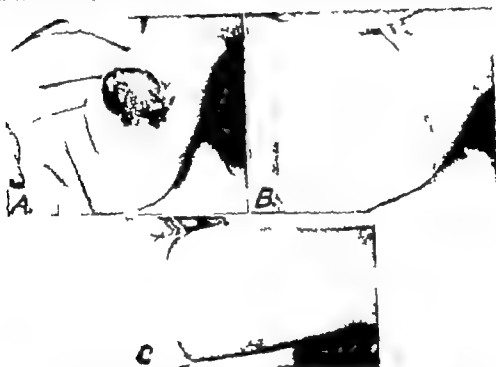


FIG. 400 Cavernous Angioma. Female, 30 eleven and one-half months. Radiation with radium and x-rays not advisable because of danger of injury to epiphyses and ovary. Under general anesthesia, the entire tumor area was removed by electrocautery and the wound sutured. (Courtesy of Dr. George E. Pflüger.)

cil of solid carbon dioxide is pressed with moderate firmness against the lesion for from twenty to forty seconds. When the pencil is removed, a deep white depression remains, which rapidly disappears as the naevus assumes its original appearance. Within twenty-four hours, a bleb develops and in a varying period of time crusting, with underlying ulceration, occurs. This is followed by healing. One or several applications,

first diluted 50 per cent with distilled water later used full strength. A 24-gauge needle attached to a 1-cc syringe, containing the sclerosing solution, is introduced with aseptic precautions, drop by drop, here and there throughout the angioma. The pain is momentary and the lesion tends to swell for a day or so, only to begin to contract later. The number of treatments will vary but can be given at two- to three-week inter-

vals until a suitable result is obtained

Radium using gamma rays only 1 c filtered through at least 0.5 mm aluminium or 2 mm of brass or their equivalent is excellent. The earlier it is used the better. *X-ray therapy* with mod-

cluded. The smallest amount of radiation should be used, with reliance on nature to do most of the work. Overdosage and beta radiation is best avoided. For surface applications, approximately one quarter to one half



Fig. 401: Cavernous Angioma. In a female, age three and one-half months. The entire nose and most of the orbit were involved, but at birth only a small, red, slightly elevated spot had been present. Surface applications of radium and later some filtered x-ray radiation caused complete disappearance of the growth. (Courtesy of Dr. George E. Pfälder.)

erate filtration also is a valuable method of treatment. For larger lesions, the x-ray treatment is preferable. For smaller lesions, radium is preferable, especially in struggling infants and when the lesions are relatively superficial. Radiation should be limited exactly to the lesion but the borders should be in

erythema dose at each sitting is usually sufficient. The dose is to be repeated at three to four week intervals. In some instances, *surgical or electrosurgical excision* and wound suture is the method of choice especially in the adult and if the lesion is on covered portions of the body or on the scalp.



Fig. 402. Cavernous Hemangioma. Female, age eight months. Cavernous hemangioma involving almost entire right side of face, neck, lip, and chin. The first lesions appeared on the lip at age of 4 to eight weeks and on cheek at age of three months. Lesion had reached present size in five months. Results shown in D and E (allowed radiation with both radium and γ -rays. (Courtesy of Dr. George E. Piller.)

GLOMUS TUMOR

This is a benign pinhead to larger sized tense or soft exquisitely tender and painful bluish red neuromyoangioma involving the glomus bodies. They may be painless however (Weidman and Wise). These bodies are present in large numbers on the extremities and are considered arteriovenous anastomoses with special nerve and muscle fibers

seated slow growing lobulated, or globular growths of purplish red, but soft, depressible and manifestly part of a deeper involvement of the vascular tree. Distortion of the area involved is usual. They may remain stationary or progress and destroy surrounding tissue.

Treatment This is often lengthy and tedious. *Sclerosing injections* given as noted under "Tuberous Angiomata" (see



Fig. 403 *Left: Lymphangioma. Not translucent nodules present since childhood.*
Right: Lymphangioma Circumscriptum.

The tumor is composed of a tangled mass of arterioles, the walls of which have epitheloid and muscle cells. Nerve fibers surround and adjoin the vascular structure. Usually one of these occurs, but more than one may do so. The commonest location is the nailbed.

Treatment *Surgical excision*

NAEVUS CAVERNOSUS (ANGIOMA CAVERNOSUM)

These appear during the first year of life. They may be part of an overlying tuberous angioma. They are deep-

p 537) and radium surface and intra-tumor implants are the methods of choice. *Plastic repair* may be deemed to be necessary after the cavernous elements have been eliminated. Pfahler has successfully used *electrosurgery* with normal suture in some cases where the lesion is supported by a good layer of subcutaneous tissue.

Lymphangioma Circumscriptum

This is a localized congenital new growth due to hypertrophy and dilation of lymph vessels.

Diagnosis and Symptoms Lymphangoma circumscriptum should be differentiated from the lymphangiectases resulting from trauma or disease, such as elephantiasis in which there is obstruction to venous or lymphatic circulation. They begin during the first year of life in the form of minute, vesicle-like, noninflammatory grouped lesions. At first they are isolated but may be so profuse and closely placed as to appear as one uniform plaque. The entire lesion may remain stationary, enlarge, and regress or undergo complete involution. In general, the plaques are rather regular rounded or oval-shaped, and appear composed of translucent lesions

covered by a smooth, thickened, or verrucose epidermis. Isolated vesiclelike lesions are almost always present at the periphery of the plaque or in several larger lesions visible at its center. When punctured, a clear fluid (lymph) begins and continues to flow for some time. Lymphangiomas may be pure, but at times show blood vascular nevus elements. Lymphangiomas are painless, and are commonly located on the upper reaches of the thighs and arms, the genital area, and the neck.

Treatment This is the same as for hemangiomas. The procedure of choice is surgical excision. Good results have been obtained with solid carbon dioxide

NAILS

General Consideration The nails are rectangular horny platelike dermal appendages resting upon the dorsal aspect of the terminal phalanges of fingers and toes. They consist of modified epithelial cells, firmly and intimately held together. Each nail rests upon a nail bed and is surrounded laterally and proximally by the nail wall, which is a fold of the epidermis. The dorsum of the nail is, convex with the base, thinner than its thickened free edge. The nail grows from the skin of the nail bed and nail wall. The visible portion of the nail is known as the body of the nail. It is usually pink because of the underlying rich vascular supply in the nail bed. The lunula is the white crescent-shaped portion of the body of the nail lying at its base. Its whiteness is due to diminished vascularity in this area of the nail bed. A nail grows its full length in about six months.

The Nails as an Aid in Diagnosis
Occupations The photographer, chemist, and dyeworker stain the nails. Yellow

staining of the nails, especially of the index and middle fingers, occurs among habitual cigarette smokers. Painters exhibit staining of the nails from various pigments. The nails become bleached in persons contacting oxidizing agents. Negroes show a blue halfmoon about the nail base instead of the usual white lunula present in the white race. The characteristic blue halfmoon of the Negro nail is said to occur in individuals possessing a distant kinship to this race. The nail bed is purple in cyanosis.

Marked blanching is often evident in anemia. Butler contends that pressure on the nail will completely exsanguinate it when the hemoglobin is less than 50 per cent as in cases of chlorosis, anemia, and severe hemorrhage. A subungual or capillary pulse is present in conditions in which the peripheral arteries are quickly filled and emptied as in aortic regurgitation. Right pressure will in this case produce alternate reddening and blanching of the nails.

The presence of a transverse nail

groove is indicative of a recent acute illness unless the groove results from traumatism. Gouty persons and those with arthritis have hard brittle, and longitudinally striated nails. Certain occupations or pursuits may cause brittle nails. Likewise the frequent use of nail polish removers or the constant exposure of nails to industrial solvents may cause the distal borders to become thin brittle and crack easily especially horizontally. These patients are often treated for suspected systemic causes when local factors are entirely operative.

Arrest in nail growth on one side of the body is indicative of hemiplegia and brain tumors on the opposite side. Comparison with the rate of growth on the sound side can be made by marking the nails with stains of silver nitrate.

Echymoses and ulcers of the nail base are present in individuals habituated to chloral. Marked sensitivity about the toenails occurs in narcotic addicts, and marks an invaluable aid in diagnosis.

White spots on the nails are indicative of mild trauma. These white spots are due to small pockets in which air accumulates between the nail and its matrix. White spots on the nails are often present in exhausting diseases, dissipation, sexual excesses, overwork, and worry.

Hangnail

The sequelae of hangnail very frequently produce considerable damage to the nail and its bed despite the fact that the condition does not involve the nail proper. Hangnail is overgrowth of the nail cuticle about the nail wall with consequent drying, cracking, and tearing. The tear is very painful and may extend into the finger for about $\frac{1}{4}$ to $\frac{3}{4}$ inch. Cracks or tears resulting from hangnails heal without much discomfort under adequate care, but infection and

consequent paronychia follow neglect.

Treatment. The prophylaxis against hangnails necessitates keeping the nail cuticle well pushed back. This is best accomplished by pushing the cuticle with *orangewood stick*, wound with cotton, after softening the nail in warm soapsuds or after applying *rosewater ointment*. Curved sharp, pointed scissors are used to trim the overgrown cuticle whenever a tendency to tearing occurs. Cuticle tears are best treated by daily applications of *tracture of iodine* working the solution under cuticle. Applications of *flexible collodion* are also curative. Scrubbing nails frequently during the day with a soft nailbrush in warm soapsuds will materially assist in the healing process and obviate infection.

Calloused Nail Groove

Calloused nail groove is characterized by callus in the lateral aspects of the nail wall. The condition is frequently seen on the large toes, and may occur on other toes and fingers. Calloused nail groove is a commonly occurring forerunner of ingrown toenails.

Etiology. The etiologic factors contributing to the formation of calloused nail groove are, (1) a natural tendency of the lateral aspect of nail to inversion with consequent irritation of the skin lining nail wall, (2) the wearing of narrow short shoes, which tends to produce irritation by forcing nail wall against the inverted nail.

The nail usually grows upward and lateralward from the corner of the toe and in this way produces a sharp point simulating a "spike." The condition is often referred to as "free nail." The nail in this way loses its natural color and luster.

Symptoms. The symptoms accompanying a calloused nail groove consist

of pain and tenderness about the corner of affected nail. Shoe pressure becomes unbearable. The mere weight of bed-clothing leads to discomfort about the affected toe. Examination reveals thickening and hardening of the nail wall. Corn formation without inflammation and suppuration may be present. Inflammation and suppuration become marked when the condition is neglected and inadequately treated. This inflammation and suppuration leads to the formation of "ingrown nails."

Treatment A calloused nail groove is treated by appropriate prophylaxis. Shoes are corrected for shape and size. The nail wall is retracted from the nail when a tendency to inversion is apparent by packing small pieces of cotton along the nail groove. *Ointments* in the form of rosewater ointment with 0.5 gm (5 grains) of ammoniated mercury to 30 gm (1 ounce) of iodine ointment, or plain petrolatum are applied to soften the skin. A piece of cotton saturated in 1 per cent solution of *lysol* or in 10 per cent solution of *potassium hydroxide* is packed into the nail corner for a period of a few minutes whenever calloused nail groove is present. The transverse portion of the nail is then removed and callus or corn carefully excised. No bleeding takes place when the line of cleavage is made between callus and underlying skin. The nail wall at site of excision is then packed and ointment applied. The patient is cautioned to report for treatment if tenderness recurs.

Ingrown Toenail

SYNONYM *Ingrown toenail*.

An ingrown toenail is one in which the nail is overlapped by the soft tissue of sides of the nail wall. Infection and granulations are present in this condition.

Etiology Ingrown toenails arise from wearing short, narrow shoes and stockings. They occur also in individuals with a natural tendency to nail inversion. Ingrown toenails may follow traumatism as in crushing injuries and in blows. The great toes are the most frequent site for ingrown nail. Other toes are not, however immune from this painful lesion. A condition similar to ingrown toenail occurs about the fingers.

Pathology Ingrown nail is a common sequela of a calloused nail groove. When an ingrown nail and a calloused nail groove are neglected and untreated, the nail penetrates the soft tissues of the nail wall and gives rise to laceration and infection. Hypertrophied soft tissues about the nail overlap the nail.

Symptoms The symptoms of ingrown nail are soreness and inflammation of the soft tissue of the distal aspect of the nail. A seropurulent discharge occurs with the formation of granulation tissue. Pain and disability usually become so intolerable that the patient cuts out a window in the shoe at the site of impingement.

Diagnosis Ingrown nail is so characteristic a condition that no difficulty arises in diagnosis. Both corners of the nail may become involved and the condition may be evident in both feet.

Prognosis The prognosis is favorable.

Treatment The treatment for ingrown nail is identical with that of calloused nail groove.

Complete removal of the nail under local or general anesthesia (pentothal sodium in particular) may be necessary in cases of recurrences. The nail is then removed in its entirety. Removal is accomplished through a curved incision into the nail wall and running around the nail from one corner to the other to

include the nail matrix proper. A periosteal elevator is employed to remove the nail matrix and adjoining soft tissue en masse. The resulting wound is then closed with sutures. A gaping wound following nail removal may occasionally persist necessitating excision of the distal phalanx.

Hypertrophy of the Nail

Nail hypertrophy is restricted to the toenails. The condition is known as "onychauxis." It involves one or more toes. The big toe is the most commonly involved digit. The nail overgrows, is irregularly thickened, assumes a green or black color and undergoes such hardening that trimming becomes difficult. The involved nail undergoes bending or curving. It simulates a claw when straight and assumes the shape of a horn when curved (onychogryphosis). Onychauxis occurs among elderly bedridden patients. Nail hypertrophy is uncommon among young people although it may occasionally be congenital. It may be associated with nerve lesions as in cases of neuritis or hemiplegia. The etiological factors in onychauxis are uncleanliness and trauma.

Treatment consists of removal of the increased nail substance.

Pachyonychia Congenita (Jadassohn)

This is one of a group of rare congenital anomalies characterized by dystrophy of the nails and apparent shortly after birth. The nails are hard, lusterless, and greatly thickened particularly at the free border.

Associated are one or several of a wide variety of apparently unrelated abnormalities of the skin and mucous membranes, among which are the following: partial or complete palmar and

plantar hyperkeratosis; hair anomalies; acroform and follicular keratosis of the type keratosis pilaris and spinulosus but suggesting Darier's disease; verrucose lesions on the knees, elbows, legs, feet, bullae often painful usually on heels and soles, and suggesting epidermolysis bullosa leukokeratosis oris in small or

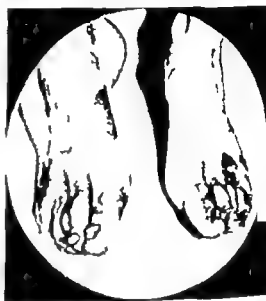


Fig. 404: Pachyonychia Congenita. (Courtesy of Dr. Carroll S. Wright.)

extensive areas (Cannon's white-sponge naevus) and extensive reticular pigmentation and atrophy of the skin (Cole) suggesting acrodermatitis chronica atrophicans.

Beau's Lines

Beau's lines are superficial depressions running transversely across the nail. They are due to a temporary interference in the nail's growth. The lines appear first at the base, gradually approaching the edge with nail growth. A period ranging from five to six months is necessary for Beau's lines to reach the nail edge. Beau's lines are ascribed to some systemic disturbance when all nails of fingers and toes are involved and to some localized cause when one or more

nails are affected. Acute infections like erysipelas, influenza, pneumonia, and vitellinosis are the most common systemic disturbances. Such conditions as severe hemorrhage following childbirth, prolonged, or severe sepsis, and nerve shock may be etiological factors. The localized appearance of Beau's lines is due to some local inflammation arising about the nail matrix as eczema, psoriasis, exfoliative dermatitis, paronychia, and trauma.

Longitudinally Striated Nails

SYNONYM *Onychorrhexis*.

So-called "longitudinally striated nails" are of common occurrence and are due to exaggerated normal longitudinal ridges in the nail bed. Marked longitudinal striation of the nail and longitudinal splitting of the nail body are present in onychorrhexis. The striations are especially marked at the edge of the nail. Longitudinally striated nails are very common in rheumatic individuals and those having foci of infection. The seat of such foci of infection may be about the teeth or about the gastrointestinal tract. Longitudinal striation of the nails may also occur in cases of myxedema, neuritis, hemiplegia, and nervous disease. It also may be associated with psoriasis, lichen planus, and eczema.

The treatment of onychorrhexis is directed to the elimination of the etiological factors.

Spoon-shaped Nail

SYNONYM *Koilonychia*.

Koilonychia is characterized by a concave nail, shaped like a spoon. The nail assumes a somewhat flattened appearance in mild cases but becomes definitely concave in the center in severe cases so that the matrix appears raised and projected around the nail wall, lift-

ing the nail off its bed. The nail surface is usually smooth and the nail substance somewhat thickened. This thickening is more marked at the margin. The common sites for spoon-shaped nails are the thumbs and forefingers, although all nails may become involved. The condition may be familial and may occur in several generations. It is common in idiopathic hypochromic anemia. A mild form of koilonychia accompanies alopecia areata. The cause is probably due to some trophic nerve lesion. It is often associated with syphilis, lichen planus, and acanthosis nigricans.

Treatment is of no avail.

Onycholysis

This is characterized by a gradual separation of the nail plate from its bed. Arteriosclerosis and hypothyroidism are common causes. Frequent contact with certain dissolved chemicals used in various industries may be a factor in some cases. It may be a factor in splitting of the nails, as may too frequent application of nail polish removers.

Leukonychia

Leukonychia is identified by white spots on the nails. The condition is common, appears punctate, and may become arranged in transverse striae. The entire nail may assume a peculiar white appearance as if it had been painted with white paint. Leukonychia can be familial and can appear in several generations. Heller ascribed it to hepatic nerve disturbance. Singer is of the opinion that it is the sign of a lowered basal metabolism. Trauma is perhaps the most common causative factor.

Onychia and Paronychia

SYNONYM *Whitlow*.

Onychia is inflammation of the nail bed, while paronychia is inflammation of

the nail wall. Onychia and paronychia occur simultaneously. Changes occurring in the nail are secondary to inflammatory conditions of the matrix.

Etiology. The infecting agents are *Staphylococci*, *Streptococci*, *B. coli*, *B. tuberculosis*, *Treponema pallidum* and yeasts.

Paronychia is more common in women than in men due probably to injury to the nail cuticle during manicuring. Cooks

whose hands are wet and whose diseased nail does not break off as in ringworm infection. Bacteriologic examination will confirm the absence of fungus.

Treatment. Paronychia is best treated by incision and drainage. Dressings of small pieces of cotton wool saturated in iodine ointment, 2 per cent aqueous solution of gentian violet or tincture of iodine with 25 per cent acetone are packed twice each day with the aid of an orangewood stick about the nail wall.



Fig. 405: Onychomycosis. (Courtesy of Dr. Jacques P. Guéquierre)

and washerwomen are commonly victims of paronychia as also are nurses and dentists. The condition may also complicate chronic eczema and dermatitis.

A chronic form of paronychia accompanies tuberculosis and syphilis. The condition seldom involves all nails. It is uncommon in the toenails. The infection spreads by contact from one nail to the other and may last for many years. The nail wall becomes red, swollen, painful and tender. Suppuration may be present.

Diagnosis. Paronychia is not to be confused with onychomycosis. Differential diagnosis is established by the characteristic lesions in the nail wall which are absent in ringworm infection. The dis-

Bier's hyperemia produced by applying a small elastic band to the base of the infected digit for half an hour morning and night, is also helpful. Roentgen-ray irradiation and autogenous vaccine are recommended methods of treatment, but must be combined with local antiseptic therapy as outlined with an attempt to reach beneath the nail wall.

Onychomadesis

SYNONYM *Defluvium ungium*.

This is characterized by the spontaneous separation of the nail from its bed. This begins at the proximal end and progresses rapidly to the free end. The cause is unknown. Familial and hereditary cases have been reported.

Onychomycosis

SYNONYMS: *Tinea ungium*, ringworm / the nails.

Onychomycosis is a fungus infection of the nails.

Etiology Onychomycosis is caused by the Epidermophyton, the Trichophyton (which is the chief offender) the Saccharomyces, and Monilia. The causative organism is determined by



FIG. 406 Onychomycosis. Causative organism is *Trichophyton interdigitale* (*T. Cyperum*). The middle finger is not involved. Note the thickening beneath the nail plate.

culture. The finding of fungi in nail paring is not easy and a careful selection of material to be examined is required.

Symptoms The clinical appearance of these infections of the nail is similar. The nail changes in fungus also closely resemble those of ringworm. The infection always begins at the free margin of the nail, and slowly undermines the nail plate until it becomes loosened and is shed or removed by trimming. The nails become yellowish or brown and are traversed by ridges and furrows. A true laric cup does not occur underneath the

nail, but a dry powder accumulates beneath its free border.

Onychomycosis results in malformation of the nail which becomes brittle, discolored, lusterless, grooved and pitted. Epidermal debris accumulates beneath its free margin. It is usually associated with paronychia inflammation (mycotic paronychia).

Masses of fungi occur in the horny stratum of the nail bed from which they grow into the nail itself.

Diagnosis Onychomycosis must be differentiated from psoriasis, eczema, pyogenic onychia and paronychia, onycholysis, pachyonychia, acrodermatitis continua, and inflammatory onychia and paronychia resulting from syphilis.

The diagnosis is made by demonstration of the causal fungi and by its clinical appearance. Subjective symptoms are usually absent.

Complications The infected nail may act as a focus and produce secondary abscesses on various parts of the body.

Prognosis This disease is very obstinate and shows no tendency to spontaneous cure. Untreated, it lasts during the patient's life.

Treatment: The affected nail should be sandpapered daily or the nail may be filed with a dental burr attached to an electric drill, until most of the infected material is removed. After the nail has been scraped, the application of 1 per cent iodine crystals in alcohol or in xylene is an effective method of treatment.

The persistent, regular and constant application of Whitfield's ointment gives highly successful results (Hodges).

Removal of the nail may be necessary and even this is not always followed by cure. The most adequate procedure for removing the nail was outlined by Walker. His procedure consists of first



Fig 407 Psoriasis. Of the nails. Not punctate pitting.



Fig 408 Epidermophytosis and Onychomycosis (thumb and small finger nails)
In same patient.

applying pieces of gauze saturated in thick zinc oxide paste to the soft tissues about the involved nail for protection. A piece of lint soaked in Fehling's solution is then placed over the nail proper and the whole digit covered with a rubber fingerstall for a period of twelve hours. The nail becomes swollen, softens, becomes gelatinous, and can readily be removed from its bed with forceps. Poultices of boracic acid and starch are then applied until the inflammation subsides.

Taylor obtained cures by ablation of the nail under procaine digital nerve block, followed by saucerization of the overlying soft tissue and careful electrocauterization of the radix to make the ablation permanent.

Iodine ointment or an aqueous solution of 2 per cent gentian violet is then applied morning and night until a new nail forms. *Röntgen-ray therapy* is valuable in some cases.

Nail Manifestations in Skin Diseases

The nails of the fingers and toes are involved in *psoriasis*. It is characterized by numerous punctiform depressions scattered over the surface of the nail, giving it the appearance of needle punctures. Scaly psoriasis may appear about the nail wall and under the nail edge, thus lifting it completely from its bed. Psoriasis of the nails has occurred without any sign of psoriasis anywhere else on the body.

The nail lesions in *dermatitis* and *eczema* vary from the mere appearance of striations and splitting to considerable distortion and exfoliation. The nails in *dermatitis exfoliativa* become thickened and present subungual hyperkeratosis. They are often shed but regenerate.

Spoon nail Beau lines, and onychorhexis may accompany *alpecia areata*.

In *primary chancre of the finger* the nail may become permanently distorted. The periungual papules occurring in *secondary syphilis* may lead to the formation of condylomatous masses around the nails, especially in the regions of the toes. The nails manifest fragility and flaking and may even be shed.

Chronic ulcerative paronychia of the toes occurs in *tertiary syphilis*. The changes may be so severe as to destroy the nails. The great toe is a common site for gumma. Children with *congenital syphilis* may show syphilitic paronychia which leads to atrophic nail or to ulcerative onychia.

A chronic form of paronychia resulting from *Mycobacterium tuberculosis* occurs frequently in children. The nail lesions of *tuberculous infection* simulate the lesions of septic paronychia. A typical smooth convex, and bulging nail, known as the Hippocratic nail, occurs in chronic pulmonary tuberculosis and in cases of chronic cardiac disease.

The nails may be shed in *scarlet fever*, *influenza pneumoniae enteric fever* and *erysipelas*. Beau's lines are often seen in these diseases. The following *skin diseases* may have associated nail manifestations: alopecia areata, pityriasis rubra pilaris, Darier's disease, ichthyosis, radiodermatitis, epidermolysis bullosa, scleroderma, sclerodactylia, poikiloderma atrophicum vasculare, leprosy, tuberculosis, and lupus erythematosus.

Newgrowths

Neoplasms do not occur on the nails. Numerous newgrowths—like chondroma, exostosis, glomus tumor or synovial cyst, fibroma wart, melanoma, and angoma—spread from the digit to the nail bed and produce changes within the nail body. Squamous-celled carcinoma may also have its origin in the nail bed.

NAPKIN ERYTHEMA

SYNONYMS: *Napkin dermatitis, erythema gluteal Jacquet dermatitis, posteriorly syphilitic*

Napkin dermatitis is a dermatitis involving the diaper regions of infants, namely the inner surfaces of the thighs, buttocks, and genitalia. It may occasionally occur on the perineum but tocks, legs and heels. It was first described by Jacquet.

Incidence *Napkin dermatitis* usually occurs among neglected infants who are subjects of malnutrition.

Etiology The condition is caused by the irritation resulting from napkins soaked in urine or the ammonia produced by certain bacilli in the feces or by the retained strong alkali soap in laundering napkins. Secondary infection may contribute to the disturbance.

Symptoms This disease is characterized by pinhead to lentil sized papules which are red or brown. The center of the lesions usually shows erosion. A certain degree of erythema is usual. Itching is rare.

Diagnosis *Napkin erythema* is often mistaken for congenital *syphilis* especially when the eruption is papular and ulcerative. The bright red lesions, the tendency to exudation, the want of configuration and definite margins, and the negative Wassermann reaction rule out *syphilis*. *Napkin erythema* is differen-

tiated from *intertrigo* by the absence of lesions on flexure surfaces. Lack of definition of lesions differentiates it from lesions arising from *fungus infection*. *Seborrheic dermatitis* simulates *intertrigo* and *napkin eruption*, but it is usually a part of a generalized *seborrheic dermatitis*.

Prophylaxis Diapers should be changed as soon as soiled. They are then soaked in a solution of boric acid for two hours, boiled, then washed in soap and water and finally rinsed with a 1:5000 solution of bichloride of mercury. Diapers should be well dried before using them again.

Treatment *Calamine liniment* alternated with a 1 per cent ointment of ammoniated mercury is an excellent medication. If the oozing is pronounced, compresses of 1 per cent solution of aluminum acetate or a 2 per cent solution of tannic acid are usually curative.

If malnutrition is present proper feeding must be instituted.

The following paste applied thickly during the day and especially at night, has been found useful:

Zinc oxide	80
Aquaphor	920
Phenol	06
Calomel	10

NECROBIOSIS LIPOIDICA DIABETICORUM

SYNONYM: *Dermatitis atrophicans lipoides diabetica*.

Necrobiosis lipoidica diabeticorum is a skin affection characterized by small circinate, oval or irregular flat, flesh colored plaques with yellowish centers.

Etiology *Necrobiosis lipoidica diabeticorum* is seen commonly among cases of diabetes mellitus, but has been observed in patients without diabetes

mellitus. Blood sugar determination and sugar tolerance tests are indicated in those without frank diabetes mellitus. It may occur also in diabetes insipidus.

Pathology The corium reveals patchy infiltration by fibroblasts and lymphocytes. Inflammation, thickening and thrombosis of the deeper vessels are



Fig. 409 Necrobiosis Lipoidica Diabeticorum. (Courtesy of Dr. Jacques P. Gueguerra.)



Fig. 410 Necrobiosis Lipoidica (dermatitis atrophicans lipoides). Note irregular plaques with well-defined borders and smooth, glistening, and waxed surfaces. Central portions of lesions are scabbed and sclerectatic. This patient is nondiabetic.

characterized by deposits of lipoids in the walls of these vessels

Symptoms The affection begins as small circinate or oval pink or yellowish nodules. The sites of predilection are the lower extremities, the tibial surfaces in particular. The upper extremity and trunk may occasionally be involved. The areas of involvement gradually en-

Telangiectasis and grayish scales may be seen on the center of plaques. Ulceration rarely occurs

Treatment The diet for diabetes mellitus is indicated for treating necrobiosis lipoidica diabetorum. The injection of insulin (3 to 5 mms of U 40) into lesions regardless of whether diabetes is present or not is helpful.



Fig 411 Necrobiosis Lipoidica. (Courtesy of Dr Carroll S. Wright)

large to form single or multiple-glazed morphealike plaques possessing yellowish centers and violaceous peripheries. The center of each plaque becomes depressed and forms an atrophic scar

An ointment of 3 per cent urea in a greaseless-cream base rubbed in at frequent intervals, is occasionally helpful

Excision and grafting may be indicated in some cases (Howard Fox)

NEUROMA CUTIS

SYNONYM *Nerve tumor*

A neuroma is a tumor composed of nonmedullated nerve fibers which are derived from the sympathetic nervous system

Incidence It occasionally follows injury. A history of heredity or of tuberculosis may be obtained

Etiology The direct cause of neu-

roma is unknown. Traumatism occurring in a tubercular patient may be the exciting factor.

Pathology The tumors consist of elastic and connective tissue mixed with nonmedullated nerve fibers which lie

tumors, the size of a pea or a bean. The tumors are sensitive, more or less painful, and are occasionally the seat of violent paroxysmal pain. The sites of predilection are the shoulders and upper and lower extremities. The lesions resemble



Fig. 412 Neuroma Cutis. I colored woman aged forty Duration fifteen years.

parallel to one another. It originates from the neurilemma. In neuroma a connective-tissue framework is the seat of medullated and nonmedullated nerve fibers together with occasional ganglionic cells.

Symptoms Neuroma cutis is characterized by discrete sharply defined, firm

keloids and nodular prurigo. They are pink or red and are firmly embedded in the derma.

Diagnosis Neuroma cutis must be differentiated from leiomyomata and the glomus tumor.

Clinically the leiomyoma is characterized by firm pink to violaceous elevated

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Clinically the *leiomyoma* is characterized by firm pink to violaceous elevated

nodules. They vary in number and are tender and usually painful. They are discrete oval and movable with the skin or form placards in which the nodules are easily palpable. They occur on the face, trunk, and extremities. The *glomus tumor* is an exceedingly tender almost always painful angioneuromuscular tumor located usually as a single lesion on an extremity. Beneath a fingernail, where its presence is indicated by a

peculiar bluish color is a favorite site.

In the final analysis, the differentiation of the three conditions depends upon the histopathologic findings.

Prognosis The tumors last throughout life and do not disappear spontaneously.

Treatment : Treatment of choice consists in *excision* under local anesthesia. *Radiation with x rays* is occasionally helpful.

ORAL DISEASES

Of Mucous Membrane

Dermatologists commonly interest themselves in diseases of the oral mucosa partly because of the frequency with which such disturbances precede, accompany or follow cutaneous diseases and partly because certain purely oral conditions closely simulate the lesions which are actually part of the dermatoses. The former have been discussed in connection with the industrial skin diseases, such as lichen planus, syphilis, pemphigus, erythema multiforme and dermatitis medicamentosa. Here will be described a number of oral mucous membrane and lingual affections.¹

Fordyce's Disease

Fordyce's disease is a symptomless condition which is characterized by flat or slightly raised, light yellow spots on the vermilion borders of the lips, on the buccal mucosa, and more rarely on the genitalia. This condition was described in a physician by Fordyce in 1896.

Incidence The lesions, or granules, are rarely seen before puberty. In the older adult group, relative degrees of involvement of the lips and oral mucosa are present in 60 to 70 per cent of the population. The disease is less commonly observed and milder in nature in the female. If mild cases are eliminated approximately 40 per cent of males and 15 per cent of females in the late adult group will show definite evidence of the disease.

Etiology Irritation due to smoking

appears to be an important predisposing cause.

Pathology The lesions of Fordyce's disease consist of hypertrophic sebaceous glands which result from irritation of the sebaceous glands caused by chewing (cheek mucosa) irritation from smoking, or from bacteria. Chambers has demonstrated by serial sections that the affected glands have an opening to the oral cavity.

The typical lesions appear as slightly raised, light yellow granules which are best demonstrated when the overlying tissues are placed under slight tension. The lesions are commonly found on the cheek mucosa in the molar region along the interdental line. They may be closely grouped about the opening of Stenson's duct or they may be diffusely distributed on the oral mucosa overlying the anterior border of the ramus of the mandible. In advanced cases, they coalesce and form yellowish, plaque-like areas.

Fordyce's granules are commonly found on the lip in cigaret smokers. The upper lip is usually more extensively involved. Persons who have thick gummy deposits on the lips and those with thick saliva seem to be particularly susceptible. At first the individual granules are visible on the vermilion border of the lip when the tissues are tensed. They appear as small orange-colored granules when lip-stick is used. As the lesions become more numerous and larger they coalesce, with a gradual disappearance of the normal red color of the vermilion lip border.

Symptoms Patients experience no symptoms with the occasional exception of a feeling of thickness of the involved tissues. They are usually unaware of the condition. Occasionally Fordyce's granules of the oral mucosa are discovered by

¹For more complete discussion of oral diseases, the following texts are recommended: (1) Burket, *Oral Medicine*. J. B. Lippincott Company Philadelphia, 1944. (2) Price, H., and Greenbaum, S. S. *Diseases of the Mouth*. Lea & Febiger Philadelphia, 1939.

the patient who will become alarmed about the nature of the lesions and require assurance of their benign nature.

Diagnosis The diagnosis of the disease can be made by inspection. On the cheek mucosa Fordyce's disease must be differentiated from early leukoplakia and the lesions of lichen planus. In early cases, the granules are best seen under a natural source of light with slight stretching of the tissues.

Treatment No treatment is indicated.

Fusospirochetal Infections

Fusospirochetal infections occur throughout the body. They can be divided into extraoral and intraoral infections. Extraoral fusospirochetal involvement of the skin, nares, conjunctivae, and middle ear is rare. Secondary fusospirochetal infections of pulmonary abscesses, tuberculous cavities, or tracheal infections may be secondary to oral infections. Fusospirochetal ulcerations of the genitalia occur in both sexes. They may or may not be associated with oral lesions. Invasion of the blood stream has also been reported. The seriousness of human tooth injuries or human bites is due to the possibility of infection with these organisms. The incidence of infection is roughly related to the cleanliness of the teeth producing the wound.

Oral fusospirochetal infections include

- (1) fusospirochetal gingivostomatitis (Vincent's infection or trench mouth)
- (2) fusospirochetal angina (Vincent's angina)

FUSOSPIROCHETAL GINGIVOSTOMATITIS

SYNONYMS *Oral fusospirochetosis, Vincent's infection, trench mouth, and necrotic or ulceromembranous stomatitis.*

The term fusospirochetal gingivostomatitis indicates the supposed etiology

and the acute inflammatory symptoms of the disease. Vincent's infection and "trench mouth" should be avoided, since patients frequently consider these "diseases" a reflection on their personal or social habits.

Incidence A few authenticated epidemics of fusospirochetal gingivostomatitis have been reported, notably the San Luis Valley (Colorado) epidemic of 1932 which affected over 3000 persons, but this disease exists mainly as isolated cases or endemic outbreaks. It is encountered in orphanages, aboard ships, in army camps, and occasionally among hospital staffs and student groups. The disease is most common in adolescents and young adults. The age incidence corresponds roughly with the eruption of the teeth, including the complete eruption of the mandibular third molars.

The disease is encountered in the summer as well as in the winter. Miller and Greenhut found the greatest prevalence in the New York area during the months of October, November, and December. It is likely to be high during and after school vacation periods. This may be related to insufficient rest, inadequate nutrition, the increased use of alcoholic beverages, or kissing.

Oral fusospirochetal stomatitis is related to the general physical status of the patient, nutritional factors, and the state of oral hygiene. While the disease is commonly found in persons with poor oral hygiene, acute fusospirochetal stomatitis may develop in those who have excellent oral hygiene and in those who do not smoke. The exact method of spread of the infection is not known. It has been suggested that it can be transmitted by direct contact, through salivary droplets, by means of contaminated eating utensils, and through infected dishwater. Regardless of the mechanism

of transmission, the resistance of the oral tissues of the host plays the dominant role in the development of the clinical disease.

Etiology The etiologic factors considered responsible for this disease include the exciting fusospirochetal organisms and numerous local and systemic predisposing factors. The predisposing factors are of major importance in determining the clinical onset of the disease. The etiologic factors responsible for the production of oral fusospirochetal infections are given below:

1. **Exciting causes.**

- a. *Borrelia vincenti*
- b. *B. melnikovi*
- c. Other members of the oral flora

2. **Predisposing factors**

Local

- (1) Erupting or malposed teeth and associated gum flaps
- (2) Poor dentistry in the form of overhanging gingival margins of fillings, ill-fitting crowns, or prosthetic appliances. Poor or inadequate contact points due to dental caries or faulty restorations
- (3) Local interference with the nutrition of the marginal and interdental gingivae due to calculus, orthodontic appliances, or metallic deposits in the tissues (bleeding stomatitis)
- (4) Areas of acetal hyperfunction, local areas of poor oral hygiene, and food impaction

Systemic

- (1) Vitamin deficiencies (C and B complex)
- (2) Gastrointestinal disturbances, chronic gallbladder disease, and intestinal hyperirritability
- (3) Chronic malnutrition and debilitating diseases
- (4) Certain blood dyscrasias, leukemia, and aplastic anemia

The fusospirochetal organisms are present in such overwhelming numbers in fusospirochetal infections of the mouth and throat that it is difficult to explain their appearance on a coincidental basis. While a few of these organisms can be

demonstrated in clinically "normal" mouths, a careful oral examination will usually reveal local areas of poor oral hygiene or a primary incubation zone, associated with a gum flap of an erupting tooth, which can explain the presence of large numbers of fusospirochetal organisms.

Under experimental conditions, the disease is not particularly contagious.



Fig. 413 Traumatic Lesion. Of the cheek mucosa due to cheek biting

Grossman and Schwartzman could not transmit the disease experimentally in children. The United States Public Health Service has observed no evidence of the contagious nature of this disease. Clinical experience however such as epidemics in fraternity houses and private and public schools suggests a contagious aspect of the disease. The British and American Navies have observed that, when facilities are not available for the sterilization of the mess utensils, the incidence of Vincent's infection is high.

Symptomatology Pain and intense burning of the oral mucosa are outstanding and universal findings in acute oral fusospirochetal gingivostomatitis. The slightest pressure on the ulcerated areas

the patient who will become alarmed about the nature of the lesions and require assurance of their benign nature.

Diagnosis The diagnosis of the disease can be made by inspection. On the cheek mucosa Fordyce's disease must be differentiated from early leukoplakia and the lesions of lichen planus. In early cases, the granules are best seen under a natural source of light with slight stretching of the tissues.

Treatment No treatment is indicated.

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- (2) fusospirochetal angina (Vincent's angina)

FUSOSPIROCHETAL GINGIVOSTOMATITIS

SYNONYMS *Oral fusospirochetal, Vincent infection, trench mouth, and necrotic or ulcer membranous stomatitis.*

The term "fusospirochetal gingivostomatitis" indicates the supposed etiology

and the acute inflammatory symptoms of the disease. "Vincent's infection" and "trench mouth" should be avoided, since patients frequently consider these "diseases" a reflection on their personal or social habits.

Incidence A few authenticated epidemics of fusospirochetal gingivostomatitis have been reported, notably the San Luis Valley (Colorado) epidemic of 1892 which affected over 3600 persons, but this disease exists mainly as isolated cases or endemic outbreaks. It is encountered in orphanages, aboard ships, in army camps, and occasionally among hospital staffs and student groups. The disease is most common in adolescents and young adults. The age incidence corresponds roughly with the eruption of the teeth, including the complete eruption of the mandibular third molars.

The disease is encountered in the summer as well as in the winter. Miller and Greenhut found the greatest prevalence in the New York area during the months of October, November and December. It is likely to be high during and after school vacation periods. This may be related to insufficient rest, inadequate nutrition, the increased use of alcoholic beverages, or kissing.

Oral fusospirochetal stomatitis is related to the general physical status of the patient, nutritional factors, and the state of oral hygiene. While the disease is commonly found in persons with poor oral hygiene, acute fusospirochetal stomatitis may develop in those who have excellent oral hygiene and in those who do not smoke. The exact method of spread of the infection is not known. It has been suggested that it can be transmitted by direct contact through salivary droplets, by means of contaminated eating utensils, and through infected dishwater. Regardless of the mechanism

of transmission, the resistance of the oral tissues of the host plays the dominant role in the development of the clinical disease.

Etiology The etiologic factors considered responsible for this disease include the exciting fusospirochetal organisms and numerous local and systemic predisposing factors. The predisposing factors are of major importance in determining the clinical onset of the disease. The etiologic factors responsible for the production of oral fusospirochetal infections are given below:

1. Exciting causes.

Borrelia vincenti.

b. B. fusiformis.

Other members of the oral flora.

2. Predisposing factors.

Local.

- (1) Erupting or malposed teeth and associated gum flaps.
- (2) Poor dentistry in the form of overhanging gingival margins of fillings ill fitting crowns, or prosthodontic appliances. Poor or inadequate contact points due to dental series or faulty restorations.
- (3) Local interference with the nutrition of the marginal and interdental gingivae due to calculus, orthodontic appliances, or sequestra deposits in the tissues (biomembrane).
- (4) Areas of archival hyperplasia, local areas of poor oral hygiene, and local infection.

b. Systemic.

- (1) Vitamin deficiencies (C and B complex).
- (2) Gastrointestinal disturbances, chronic gallbladder disease, and intestinal hyperirritability.
- (3) Chronic malnutrition and debilitating diseases.
- (4) Certain blood dyscrasias, leukemia, and aplastic anemia.

The fusospirochetal organisms are present in such overwhelming numbers in fusospirochetal infections of the mouth and throat that it is difficult to explain their appearance on a coincidental basis. While a few of these organisms can be

demonstrated in clinically "normal" mouths, a careful oral examination will usually reveal local areas of poor oral hygiene or a primary incubation zone associated with a gum flap of an erupting tooth which can explain the presence of large numbers of fusospirochetal organisms.

Under experimental conditions the disease is not particularly contagious.



Fig. 413. Traumatic lesion. Of the cheek mucosa due to cheek biting.

Grossman and Schwartzman could not transmit the disease experimentally in children. The United States Public Health Service has observed no evidence of the contagious nature of this disease. Clinical experience however such as epidemics in fraternity houses and private and public schools suggests a contagious aspect of the disease. The British and American Navies have observed that when facilities are not available for the sterilization of the mess utensils, the incidence of Vincent's infection is high.

Symptomatology Pain and intense burning of the oral mucosa are outstanding and universal findings in acute oral fusospirochetal gingivostomatitis. The slightest pressure on the ulcerated areas

the patient who will become alarmed about the nature of the lesions and require assurance of their benign nature.

Diagnosis The diagnosis of the disease can be made by inspection. On the cheek mucosa Fordyce's disease must be differentiated from early leukoplakia and the lesions of lichen planus. In early cases the granules are best seen under a natural source of light with slight stretching of the tissues.

Treatment No treatment is indicated.

Fusospirochetal Infections

Fusospirochetal infections occur throughout the body. They can be divided into extraoral and intraoral infections. Extraoral fusospirochetal involvement of the skin, nares, conjunctivae and middle ear is rare. Secondary fusospirochetal infections of pulmonary abscesses, tuberculous cavities or tracheal infections may be secondary to oral infections. Fusospirochetal ulcerations of the genitalia occur in both sexes. They may or may not be associated with oral lesions. Invasion of the blood stream has also been reported. The seriousness of human tooth injuries or human bites is due to the possibility of infection with these organisms. The incidence of infection is roughly related to the cleanliness of the teeth producing the wound.

Oral fusospirochetal infections include

- (1) fusospirochetal gingivostomatitis (Vincent's infection or trench mouth)
- (2) fusospirochetal angina (Vincent's angina)

FUSOSPIROCHETAL GINGIVOSTOMATITIS

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chetal infection secondary to bismuth or mercury intoxication.

The heavy metallic odor which is associated with oral fusospirochetal infection—or cultures of these organisms—is so characteristic that once experienced it is seldom mistaken. Prinz believes that it is the outstanding symptom of the acute disease.

In all cases of oral fusospirochetal infection, the tonsils should be carefully

The patient may complain of weakness due partly to the infection and partly to the decreased food intake. Headache, mild backache, joint pains, and general malaise are common. The patient does not obtain the usual pleasure from smoking. In children, temperatures of 103° F (39.4° C) are not unusual, and temperatures as high as 104° F (40° C) have been reported in adults; but since most of these cases terminated fatally the hy-

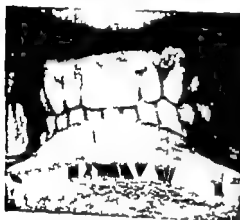


Fig. 415 Fusospirochetal Gingivitis. *Left* Early stage with beginning ulceration of the interdental gum tissue between the upper left second incisor and canine. *Right* Acute case treated for several months with repeated applications of 10 per cent chromic acid. Note the extensive tissue destruction and bleeding of the enamel.

examined for lesions which may serve as primary incubation zones. If present, both sites of the disease must be treated simultaneously. The regional lymph nodes are usually enlarged, if not visibly so, at least palpably. The lymphadenopathy is variable, depending on the individual, the severity of the disease, and the amount of secondary infection. Adenopathy is more common in children and in cases of circumcoronal infections about erupting third molars.

Constitutional symptoms are of little importance in adults, but they are more common and severe in children. Occasionally however adults are markedly prostrated and require hospitalization.

perpyrexia may have been due to secondary factors.

Laboratory Aids to Diagnosis The bacterial smear is an important aid in the diagnosis of fusospirochetal stomatitis. It is most useful in the subacute and chronic forms of the disease, and it furnishes important information of the progress of treatment. The material for the smear must be taken from the interdental ulcerated areas, the gum tissues adjacent to the erupting teeth or other primary incubation zones. The smears can be stained with a saturated solution of crystal violet. The finding of an occasional organism is of little significance; they must be present in overwhelming

is exquisitely painful and even speaking and the passage of cold air or food over the affected tissues are distressing. These symptoms prevent rest eating and the maintenance of oral hygiene.

Sialorrhea is a constant symptom. The expectorated saliva is frequently bloody from the bleeding ulcerations. Spontaneous gingival hemorrhages, which occur during sleep are common symptoms in children. A metallic taste and a peculiar odor to the breath are noted by some

The ulcerations are found on the palate on the inner surfaces of the cheeks, and on the lips in severe cases. The palatal ulcerations develop by direct extension of the gingival lesions, while the buccal and labial lesions result from contact of these tissues with the gingival ulcerations. A yellowish or grayish white pseudomembrane is present in many cases. It is removed with comparative ease leaving a cup-shaped bright red ulceration which bleeds readily. The

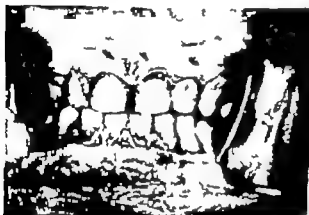


Fig. 414: Fusospirochetal Gingivitis. *Left:* Acute case in a twelve-year-old girl with ulceration and pseudomembrane formation along the marginal gingivae. Moderate elevation of temperature. Hematologic findings normal. *Right:* Fusospirochetal ulceration of the palate associated with fusospirochetal gingivitis.

patients. The teeth are frequently elongated and have a "woody" feel due to involvement of the periodontal membrane.

The ulcerative and membranous lesions are the characteristic objective findings. The fusospirochetal ulcerations appear as small punched-out lesions that develop first on the interdental papillae and the marginal gingivae. Frequently all that remains of the interdental papilla is a small triangular shaped mass of necrotic tissue. The ulcerative lesions progress rapidly and it is not unusual for an interdental papilla to be destroyed in six to eight hours. Acute pain and bleeding result from the slightest pressure on these areas.

punched-out characteristic of the ulcers is seen to best advantage on the interdental gum tissue and on the palate. If the ulcerative process continues, as it may in untreated cases, the alveolar process becomes exposed and the teeth and bone are sequestered.

The teeth and gingival tissues may be stained a reddish brown from the decomposing blood. The tongue is usually coated due to neglected oral hygiene or treatment with oxygen liberating agents. The contact ulcers which develop occasionally on the tongue are exquisitely painful. The saliva is thick, viscid, and increased in amount. The salivary glands are often sore and painful. This is particularly true in cases of oral fusospiro-

for the tubercle bacillus in only a small proportion of the cases. Complete physical and roentgenologic examinations, biopsy studies, and guinea pig injections may be required to establish a diagnosis.

The lesions of acute generalized syphilis should not be confused with those of fusospirochetal infection. Mucous patches are uncommon on the gingival tissues, and they are not as painful as the lesions of fusospirochetal stomatitis. They do not result in the marked tissue de-

structive symptoms without eliminating the predisposing factors, resulting in frequent recurrences or the persistence of a chronic infection which may produce considerable tissue destruction. If the deeper tissues are involved such as they may be in fusospirochetal angina, intravenous or surgical therapy is indicated.

Since dental operations are required in almost all cases to eliminate the predisposing causes of the disease and since these causes are most important in the

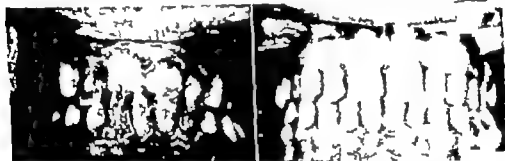


Fig. 416 Fusospirochetal Stomatitis. Before (left) and after (right) photographs of patient with severe form of disease. Time interval ten days. Treated by general oral hygienic measures and the topical application of noncaustic medications. Note the preservation of the gingival tissues.

struction. The mucous patches are slightly raised and have a typical translucent coating.

Most of the uncommon lesions of the oral mucosa are first diagnosed as a fusospirochetal infection. This is due to the lack of familiarity with the typical clinical features of fusospirochetal disease in the mouth and the failure to consider that oral lesions may represent a manifestation of some systemic disease such as tuberculosis, leukemia, or syphilis.

Treatment The treatment of oral fusospirochetal stomatitis is directed towards eliminating the painful subjective symptoms and the predisposing causes of the disease. The use of topical medication alone or intravenous surgical therapy only ameliorates the subjec-

clinical production of the disease and its recurrences, the treatment of fusospirochetal infection should be the responsibility of the dentist. Prompt, intensive, conservative local therapy will give satisfactory clinical results with fewer recurrences and the least amount of tissue destruction.

The general objectives in the treatment of oral fusospirochetal infections are: (1) the reduction of the numbers and possibly the virulence of the oral bacteria through mechanical means such as mouth washes, douches, and topical medication; (2) the elimination or correction of as many of the local and general predisposing factors as is possible; and (3) measures taken to increase local and general tissue resistance including the estab-

numbers to constitute a positive smear.

The blood count is of little positive value in the diagnosis of fusospirochetal infections although it will eliminate the possibility of the oral lesions being secondary to leukemia, malignant neutropenia or aplastic anemia. A blood count should be taken on all cases of acute ulcerative lesions of the mouth if there is any doubt that the condition is a fusospirochetal infection or if the lesions fail to respond promptly to local treatment.

Diagnosis. The sudden development of punched-out ulcerations on the marginal gingivae or the interdental papillae with acute pain and hemorrhage from the slightest pressure on these lesions, sialorrhea and a metallic odor with comparatively mild systemic symptoms should suggest a diagnosis of fusospirochetal infection. The clinical impression should be corroborated by bacteriologic smears. In chronic infections, where clinical symptoms are less striking, the disease is often unrecognized unless smears are routine.

Differential Diagnosis. The conditions most likely to be confused with fusospirochetal infections are herpetic and streptococcal stomatitis, the oral lesions of malignant neutropenia, leukemia, tuberculosis, and syphilis.

Herpetic stomatitis should cause little difficulty in diagnosis. The premonitory symptoms in this disease, the development of the lesions on the smooth mucosal surfaces, their common occurrence on the vermillion border of the lips, and the yellow opaque appearance of the healing vesicles should permit a diagnosis of herpetic stomatitis. The oral lesions of erythema multiforme also have a predilection for the smooth mucosal surfaces, and they have little tendency to ulcerate. Both of these diseases lack the characteristic metallic odor which is typical of fusospirochetal stomatitis.

In streptococcal stomatitis, the smooth mucosal surfaces of the cheeks, the alveolar gingivae, and the palate are involved. The eroded areas are not found on the marginal gum tissue and ulceration is rarely present. The diffuse nature of the involvement, the increase in the systemic response, and the bacterial smear findings permit a differential diagnosis. Healing herpetic lesions of the oral cavity should cause no difficulty in diagnosis.

The sudden onset and the similar appearance of the lesions of malignant neutropenia and fusospirochetal infections may present a serious diagnostic problem. In malignant neutropenia, the ulcerations are usually less painful, and they do not elicit an acute inflammatory response. The base of the ulcer is greenish black. Bacterial smears are of little value. The blood count will establish the diagnosis. In the gingival lesions associated with leukemia, there is a more diffuse hypertrophy of the gums, the mouth odor is frankly putrid in nature, and extensive sloughs or ulcerated areas are frequently present.

Tuberculous lesions of the oral mucosa are frequently diagnosed as fusospirochetal lesions because of the intense pain and the marked salivation which is associated with them. Oral tuberculous lesions are found chiefly on the lateral or central portion of the dorsum of the tongue, the commissures of the mouth, the cheeks, or the palate. They are uncommon on the gingiva. Oral tuberculous lesions are aggravated by intravenous arsenical or bismuth therapy. The lack of acute inflammatory response, in spite of the acute pain which the patient experiences, the sluggish nature of the lesions, and the patient's history will assist in making a tentative diagnosis. Direct smears from the lesions are positive

tra for the treatment of fusospirochetal stomatitis have appeared. The results suggest that this drug will control the acute bacterial phase of the infection, but recurrences are common unless the local predisposing factors are eliminated by instrumentation and dental treatment. In deep infections of the tissues, this type of treatment should be tried.

Leukoplakia

Leukoplakia is a chronic disease characterized by a loss of translucency thickening and hyperkeratinization of the mucosal tissues. While leukoplakia is found in the trachea renal pelvis, bladder and cervix uteri, the discussion which follows is primarily concerned with the oral involvement. *Leukoplakia buccalis*



Fig. 417 Left Leukoplakia. Right Leukoplakia and Syphilis.

The fact that Vincent's infection develops in patients receiving intravenous arsenical therapy for syphilis led Loeckwick to investigate further the value of this type of therapy in the treatment of Vincent's infection. Even the intensive five-day treatment of syphilis did not result in negative smears for the oral fusospirochetal organisms. Bismuth therapy neither prevents the development of fusospirochetal stomatitis nor is it effective in its treatment. Under ordinary circumstances, the most satisfactory results will be obtained if the responsibility for the treatment of this disease is delegated to the dentist.

is important because of the lack of subjective symptoms and the secondary malignant changes which may develop.

Incidence. Leukoplakia is a disease of middle life which is found almost exclusively in the male sex. While the exact causative agent has not been determined much is known about the etiology of this disease. Among the general predisposing factors to be considered are the following: (1) racial or constitutional types; (2) hormonal or nutritional disturbances; and (3) the existence of certain systemic diseases. Multiple causative factors are usually present in any case. Definite evidence of the causa-

lishment of good oral hygiene. Fusospirochetal infection should not be considered as cured until all the predisposing factors have been eliminated.

In acute infections several general measures are recommended. *Rest* preferably in bed is desirable and beneficial. Smoking and alcoholic beverages are absolutely prohibited. Highly spiced and seasoned foods should be avoided. A mild saline cathartic can be administered. The diet should be semisolid, bland and nutritious and it should contain an abundance of protective foods. If citrus fruit juices cause undue pain the synthetic ascorbic acid 300 to 500 mg per day can be administered. Therapeutic doses of the entire B complex are also desirable. While vitamin C has no specific curative effect it seems to augment the usual local treatment probably by its beneficial effect on wound healing. Nicotinic acid is not of specific curative value except in those patients where the fusospirochetal ulcerations are secondary to a nutritional deficiency. In primary fusospirochetal gingivostomatitis in adults the disease does not respond favorably to nicotinic acid therapy alone, even in doses as high as 1000 mg per day. If the fusospirochetal infection is secondary to heavy metal therapy it may be necessary to stop this during the early stages of the treatment.

The frequent use of hot saline mouth douches or 1 1/4 per cent hydrogen peroxide mouth washes is beneficial. Sodium perborate should not be used because of its caustic and irritating action. In addition it may cause undesirable hypertrophy of the filiform papillae of the tongue. Gluckman and Bibby studied the effect of repeated applications of sodium perborate, flavored sodium perborate and "neutralized" sodium perborate (neutrox) pastes on the gingival mucosa

of dogs. All these preparations cause inflammation, edema or ulceration with considerable variation in response in individual dogs.

The topical application of drugs will relieve the acute painful symptoms. The areas of ulceration should be cleansed of debris by means of cotton swabs dipped in 3 per cent peroxide before the topical application of any medication. Tincture metaphen 1:200 (untinted) trogen,* 10 per cent sodium carbonate and 10 per cent aqueous arsenphenamine are excellent topical medicaments. They will control the painful symptoms and will not cause tissue damage like the caustic drugs. A thick, creamy aqueous paste of zinc peroxide is excellent and it can be used at home by the patient. It is the best form of local treatment for the acute metallic stomatitis. In the case of fusospirochetal infections associated with third molar flaps, zinc peroxide, eugenol and olive oil in equal parts are effective agents. These drugs will control the acute painful symptoms, after which the predisposing causes of the disease can be eliminated and the patient instructed in proper oral hygiene by the dentist.

The local use of the various sulfonamides has been advocated for the treatment of fusospirochetal stomatitis. Chewing gum medicated with sulfathiazole or the sucking of sulfonamide tablets has been suggested. In the acute form of the disease, the oral tissues are too painful to permit chewing and in the less painful stages of the disease, other agents are as effective. Rosenthal has shown that sulfadiazine is the most active sulfonamide against the oral spirochetes.

A few reports dealing with both the topical and intramuscular use of penicillin

Crystal violet
Brilliant green
Ethyl alcohol (90%) q.s.

10
1.0
100.0



Fig. 418 Leukoplakia. *Left* Typical location and appearance of leukoplakia buccalis. The patient is 52 years of age. He is heavy pipe smoker. *Right* Extensive leukoplakia of the tongue in Negro. Blood-Wassermann reaction negative



Fig. 419 Leukoplakia. Localized areas developing about the inflamed orifices of the parotid glands. The patient was heavy smoker

tive role of any one factor or the relative importance of a single factor is not available

Blond-skinned blue-eyed, and lightly pigmented persons are more susceptible to leukoplakia. The disease is said to be less common in the Negro. There is some evidence that vitamin A deficiency may be a predisposing factor and Koop stressed the role of B-complex deficiency in the causation of this disease. Judged by the therapeutic response following vitamin therapy vitamin deficiencies are not important general predisposing factors. Nathanson and Weisberger suggested that hormonal disturbances may play a role in its development.

The associated occurrence of syphilis and leukoplakia has been recognized for many years, and some clinicians have considered syphilitic hyperkeratosis and leukoplakia identical. Leukoplakia is associated with syphilis but there is no scientific basis for regarding one as the result or the cause of the other. Eichenlaub studied the incidence of leukoplakia, syphilis, and tuberculosis in a large number of World War I veterans. These findings are summarized as follows:

LEUKOPLAKIA BUCCALIS, SYPHILIS AND TUBERCULOSIS IN WORLD WAR I VETERANS

	Number	Number with Leuko- plakia	Per Cent Involved
Syphilis	623	83	5.3
Tuberculosis	2408	39	1.6

While the number of patients with syphilis and tuberculosis is not comparable, there is a slightly increased percentage of leukoplakia in the syphilitic patients (5.3 per cent) as compared with the percentage of leukoplakia in the tuberculous patients (1.6 per cent).

Etiology Local predisposing factors include traumatic, chemical (perhaps electrogalvanic) thermal, and bacterial forms of irritation. Mild traumatic irritation of long duration due to a broken tooth, a faulty crown, malposed teeth, fractured dentures, or abnormal habits are local predisposing causes. Fredell and Rosenthal stressed the etiologic role of chewing tobacco in the production of leukoplakia and carcinoma of the mouth. Tobacco particularly when it is smoked, is an important predisposing factor. It has been the author's experience that leukoplakia is more commonly found in persons who smoke a pipe or cigars excessively. Prins stated that the mordanting and flavoring agents which are used in the curing of tobacco are the important irritating factors. The distillation compounds which are formed during the combustion of tobacco have also been incriminated. Roffo has succeeded in the experimental production of leukoplakia by blowing tobacco smoke through a tube on the oral mucosa of rabbits. A more diffuse application of tobacco, or tobacco smoke did not produce leukoplakia.

Roffo also demonstrated that different distillate products of tobacco have different irritating effects. The watery distillate, 212 to 248° F (100 to 120° C) was relatively innocuous to the ears of rabbits, while a thicker liquid, 248 to 302° F (120 to 350° C) and the residue resulted in the development of carcinoma in 85 per cent and 70 per cent respectively under the same experimental conditions. The same author and also Ullman have noted high blood cholesterol levels in patients with leukoplakia. This substance can be demonstrated at times in histologic specimens of the lesions.

Other forms of irritation in the form of highly spiced foods, alcoholic beverages,

develop where the quid is customarily held.

The usual location of leukoplakia is on the cheek mucosa at the oral commissure extending posteriorly on this tissue on a line parallel to the occlusal plane of the teeth. The concentration of soluble irritating products in the saliva in this area resulting from the smoking or chewing of tobacco, is believed to account for

plakia which surround the reddened and inflamed orifices of the ducts. When the lumen is obstructed, the palatal glands become enlarged and form multiple nodular elevations beneath the leukoplakia on the palate. In advanced cases, the entire hard palate is involved. In denture-wearing patients, leukoplakia is frequently found just beyond the margin of the denture. The denture apparently



Fig. 421 Leukoplakia. Granular form associated with carcinoma of the right alveolar ridge. The growth had already involved the maxillary sinus.

the frequent occurrence of leukoplakia in this region. The dorsum of the tongue is another common site, particularly in those cases where syphilis is a predisposing factor. The mobility of this organ may be noticeably impaired, particularly when there is an associated syphilitic sclerous glossitis. The sublingual space and the buccal and labial alveolar gingivae are involved less frequently.

Leukoplakia of the palate presents a variety of clinical appearances. The hyperkeratinization frequently develops first about the openings of the ducts of the small palatal glands, giving rise to yellowish-white, circular areas of leuko-

protected the supporting tissues from the local irritating factors—usually the products of tobacco combustion.

There is no lymphadenopathy in uncomplicated leukoplakia. If cracking, fissuring, or ulceration is present with secondary infection, there is usually some enlargement of the lymph nodes. If hard shotty lymphadenopathy is associated with advanced leukoplakia, the possibility of malignant change with regional metastasis should be considered.

Some clinicians grade leukoplakia numerically (Grade I, II, III, and IV) according to the severity of the disease. This practice is of questionable clinical

ages, and betel or areca nut chewing are possible irritating factors. Electrogluvanism is of little clinical importance in the causation of leukoplakia. Bacterial infection is an aggravating factor in leukoplakia if not a predisposing cause. While leukoplakia develops frequently in persons with good oral hygiene, it is more often associated with poor oral hygiene and poor dental conditions. Leukoplakia

opacity and thickening of the epithelial tissues comprise the typical irregularly outlined, yellowish white patch of leukoplakia. In the early stages, leukoplakia is not a permanent lesion and the abnormal keratinized epithelium may be replaced by normal tissues with the disappearance of the lesion.

The typical patch of leukoplakia has well-defined margins, and there is a loss



Fig. 420: Leukoplakia

is found commonly in edentulous patients. It is not uncommon to observe leukoplakia developing in denture-wearing patients beyond the margin of the denture.

Symptomatology The lack of subjective symptoms is one of the characteristic features of early leukoplakia. It is not unusual for advanced lesions to develop without giving rise to any symptoms. In the initial stages there is dryness, erythema, and slight roughening of the mucosal tissues. These changes are usually ascribed to smoking or some form of mechanical irritation. A whitish

of flexibility or mobility of the involved tissues. As the lesion progresses, cracking and fissuring may develop with bleeding, secondary bacterial infection, and pain. When these symptoms develop, they are suggestive of malignant change.

The location of the leukoplakic patches is an important aid in diagnosis. The shape and the extent of the lesions are modified to some degree by the predisposing factors. In pipe smokers, the leukoplakia develops commonly where the end of the pipe stem comes in contact with the tongue or the cheek mucosa. In tobacco or snuff chewers, the lesion may

tion of the dorsum of the tongue they have a blue color and they also lack the induration exhibited by the lesions of leukoplakia.

Traumatic lesions of the cheek mucosa such as those resulting from cheek biting, develop on the cheek on a line parallel with the occlusal plane. Lesions are

formed plainly of the possibility of malignant change if the treatment advised is not followed explicitly. Clinical improvement should not be anticipated in terms of weeks but rather in terms of months.

Conservative treatment consists of complete cessation of smoking, the use of



Fig. 423 Leukoplakia. Undergoing carcinomatous degeneration in the palatal region.

frequently seen at right angles to this main line where the tissues have been traumatized by individual teeth. The distribution of the lesions with their indefinite margins, their lack of induration, and the macerated appearance permits differentiation from the lesions of leukoplakia.

Treatment Early leukoplakia can be treated satisfactorily in the cooperative patient in most instances by conservative measures. The patient should be in

alcohol and the eating of highly seasoned and hot foods. All forms of local irritation should be eliminated by appropriate dental treatment. Marked improvement will frequently take place if the lesion is rubbed gently five minutes twice daily with a soft cloth moistened with water and castile soap. It is not unusual to observe leathery areas of leukoplakia become softened under this form of treatment and disappear completely. Cauterizing agents are of no value and they may be

value. Benign appearing lesions may progress in spite of conservative treatment, while advanced lesions often regress under a similar type of treatment. In general the verrucous and fungating forms are less dangerous than the smoother plaque-like types which have a greater tendency to undergo malignant change. Any area of leukoplakia with

also more imperative. Biopsy study is indicated when malignant degeneration is suspected. If the lesion is small, it should be removed in its entirety when the biopsy specimen is secured.

Lichen planus of the cheek consists of linear interlacing lesions which develop first and most extensively in the molar region. They tend to fan out and disap-



Fig. 422. Leukoplakia. Showing beginning malignancy change.

cracking and fissuring should be regarded as malignant until proven otherwise.

Diagnosis. Leukoplakia can be diagnosed in most instances on the clinical appearance and distribution of the lesions in association with one or more general or local predisposing factors. Carcinoma, lichen planus, traumatic lesions of the oral mucosa, geographic tongue and thrush must be considered in the differential diagnosis. When fissuring or ulcerative changes are present the correct diagnosis becomes more difficult and

appear as the oral commissure is reached. They have a bluish purple color. The margins of the lesions are not as clearly demarcated from the normal mucosa, and they are not indurated as are the lesions of leukoplakia. Except in the erosive type of lichen planus, the superficial mucosal surface is smooth and presents little change. Lichen planus of the tongue has a plaque-like distribution which must be differentiated from leukoplakia of this organ. The lesions of lichen planus appear characteristically in the central por-

the supporting tissues because of resorption of the latter a small fold of soft tissue becomes pinched between the denture flange and the bony supporting area. In time this results in a decubital ulceration or a fissured epulis.

The simple decubital ulcer will rarely present any diagnostic problem. When there is fibrous hyperplasia with a central linear ulceration and associated edema of the surrounding tissues, the lesion assumes many of the characteristics of a

to small, firm, nonpainful, nodular elevations resulting from the hypertrophy of the small mucous glands of the palate. The bleeding, edematous tissue reaction as well as the nodular lesions are frequently diagnosed as malignant growths. The distribution of the lesions to an area on the palate which corresponds to the relief chamber will permit a correct diagnosis. In most cases, the lesions will disappear when a satisfactorily fitting denture is made.



FIG. 424 Oral Lesions. Left Hypertrophic tissue changes of the palate due to ill-fitting denture with well-defined relief or "suction" chamber. Right Fissured areas of hyperplastic tissue associated with an ill-fitting denture.

neoplastic growth. In a granuloma fissuratum, the ulcerated area is definitely localized to the tissues in contact with the flange of the denture, and the ulcerated area is cleaner than that found in a malignant growth.

Ill-fitting dentures with an accentuated relief or suction chamber will occasionally give rise to changes in the tissues underlying the suction chamber. These are secondary to atmospheric pressure changes which are produced by the slight movement of the denture during chewing. The tissue reaction varies from reddened, edematous, hypertrophied tissue

Allergy to the denture base material may be a cause of diffuse stomatitis or actual, superficial, necrotizing lesions of the denture-bearing and contacting tissues. Allergy to acrylic denture-base material is uncommon, and, when found, it is usually due to the use of materials which are not purified sufficiently for use in the mouth. Irritating impurities in the monomer are usually the sensitizing agents.

Patch-testing of scrapings from processed acrylic denture or of the monomer and polymer will establish the diagnosis. While the patch-testing can be performed

harmful Sutton and Sutton state that "applications of silver nitrate are particularly successful in promoting the progress of leukoplakia into carcinoma."

Nathanson and Weisberger reported satisfactory clinical response following the injection of female sex hormones. Garb and Rubin also noted involution of leukoplakic patches of the tongue and cheek following ten intramuscular injections of 10 mg. of *testosterone propionate*. After 330 mg. of testosterone had been injected the tissues were free from leukoplakic spots. There was a partial recurrence of the lesions following a lapse of treatment of three months. Male or female sex hormone therapy has not been employed in any large-scale study.

Large doses of vitamin A have been used with occasional beneficial results. Koop reported the successful use of vitamin B complex in the treatment of oral leukoplakia. The clinical response following vitamin therapy has not been encouraging.

The advisability of placing dentures over areas of leukoplakia arises frequently in dental practice. Vaughn has studied the effect of denture wearing on leukoplakia of the hard palate. He noted that, if the denture is properly constructed (not itself a source of irritation) it may constitute a good form of treatment. Leukoplakia was observed to disappear completely under properly constructed dentures in a relatively short time when there was no change in the personal habits of the patient. The insulating and protective functions of the denture were believed responsible for the clinical improvement. The time for response varied from a few weeks to months.

In advanced lesions, where the leukoplakia is localized surgery or electrocautery may be indicated. When secondary

malignant degeneration has occurred, radiation therapy with or without surgery should be employed. Friedell and Rosenthal obtained good results from radiation therapy alone.

Prognosis The prognosis in early cases of leukoplakia is good if the patient cooperates. Sturgis and Lund found that 12 per cent of a group of 296 patients with leukoplakia later developed cancer. In some cases the lesions will progress in spite of all forms of treatment; and in other instances where advanced lesions are present they will regress under conservative treatment.

If verrucous changes occur or if persistent erosion or fissure develops, electrodesiccation is advisable. As a rule, electrodesiccation of large areas of leukoplakia is apt to be followed by recurrences.

Oral Lesions Associated with the Wearing of Dentures

These lesions can be divided for convenience into those due (1) to the flanges of ill fitting dentures, (2) to accentuated suction or relief chambers, and (3) to allergy to a denture-base material.

Along the periphery of an ill-fitting denture, two types of tissue reaction may be noted: (1) a decubital ulceration (prosthetic ulcer) or (2) a mass of hypertrophied or hyperplastic tissue, presenting a deep fissure which may or may not be ulcerated (the fissured epulis or granuloma fissuratum). These lesions are distinct from the acute, painful, mechanical irritative lesions which are frequently experienced following the insertion of a new denture. They are found commonly in the canine area of the maxilla and the incisor and canine regions in the mandible. They are rarely seen on the lingual side of the alveolar ridge. When a space develops between the denture flange and

An irregular grooving in the dorsum of the tongue is frequently seen. When these grooves extend to a depth of 3 to 4 mm., they produce a grooved or scrotal tongue. While this condition was, at one time, associated with syphilis, there is no basis for this view. A grooved tongue may exist as a familial trait, but



Fig. 426 Strawberry Tongue. Result of bismuth therapy for syphilis.

it occurs in most instances as an isolated developmental anomaly.

When deep grooves are present, the bacteria and accumulated debris in the depth of the grooves may give rise to a mild inflammatory reaction of the adjacent tissues with swelling. Treatment consists of manipulating the tongue so that the fissures can be cleansed mechanically with cotton swabs and a mild antiseptic agent, such as 1 per cent crystal violet, applied to the inflamed areas.

Glossitis Rhombica Mediana

Glossitis rhombica mediana is manifested clinically by a raised, diamond-shaped mass of smooth or nodular tissue

which is situated anterior to the foramen cecum. This mass of tissue does not have the usual papillary coating of the dorsum of the tongue. The lesion represents a developmental anomaly arising from the lack of fusion of the lateral halves of the tongue prior to the upward growth of the tuberculum impar.

When deep grooves are present, there may be secondary inflammatory changes. Many times the patient will become alarmed upon seeing this tissue mass for the first time. This lesion is important mainly for the diagnostic problems which it provokes and not because of its symptomatology or sequelae. Malignant changes have not been reported arising from this tissue. No treatment other than assuring the patient of the benign nature of the lesion is indicated.

Black Tongue

Black tongue is a recognized clinical entity which results from the growth of a pigment producing micro-organism on the hyperkeratinized, hypertrophied filiform papillae of the dorsum of the tongue. It is usually seen in the male. This disease is of greater academic interest than it is clinical significance.

Etiology There is no general agreement as to the micro-organism responsible for the black pigmentation. Weidman has called attention to the possible relation between trichomycosis and black tongue. The *Streptothrix* thread forms which he isolated from a case of black tongue produced typical lesions of trichomycosis in a monkey. Yates found *Torula* present in two cases of black tongue which he studied. Monilia organisms have been recovered repeatedly but they are probably coincidental rather than causative.

There are numerous reports where the patient with black tongue had placed a cigaret he was smoking on a piece of

on the arm it is desirable to perform it on the oral mucosa using the method described by Goldman and Goldman. Kaminsky and coworkers have presented evidence which indicates that many of the cases of so-called "denture sore mouth" are allergic manifestations to some ingredient in the vulcanized denture.

Allergy due to metallic denture bases, particularly those which contain cobalt

atrophy of the papillary coating of the tongue. Waldenstrom has shown that any process which interferes with cellular respiration of these structures may give rise to papillary atrophy. Atrophic tongue changes which are not commensurate with the patient's age are usually indicative of a more profound systemic disturbance and in some instances, they are of specific diagnostic significance. The tongue furnishes considerable in-



Fig. 420: Abnormally Coated Tongue. With secondary discoloration of the elongated filiform papillae. No pigment producing micro-organisms were isolated from the lesion. Pseudo-black tongue.

or nickel have also been reported. They are of little clinical importance because of their rarity.

Tongue

There are few true diseases of the tongue. Our knowledge about pathologic changes in this organ and their systemic significance, is far from complete. It is now recognized that a coated tongue is of limited clinical significance. It represents a condition of altered physiology of the oral cavity which prevents normal wearing away of the papillary coating or an abnormal stimulation of the papillary growth due to some drug such as sodium perborate.

Numerous diseases are associated with

formation as to the general state of body hydration.

The tongue is also a frequent site for the lesions of widespread systemic diseases such as syphilis, tuberculosis, lichen planus, herpes simplex, erythema multiforme and the various drug eruptions. Leukoplakia of the tongue is both a common and an important disease.

Several diseases, or affections, of the tongue merit a separate description.

Scrotal Tongue

A certain amount of shallow grooving of the tongue is commonly seen particularly on the dorsum of the tongue along the line of fusion of the lateral halves.

Geographic Tongue

SYNONYMS *Erythema migrans*,
wandering rash of the tongue.

Etiology Geographic tongue is a common affection of the tongue of unknown causation. It is of little clinical significance. It is found most frequently in the adolescent and young adult with some predisposition for the female sex. Persons with an allergic background and those with nervous tendencies are com-

The lesions are found on the dorsum and lateral margins of the tongue. They consist of a central area where the papillae are absent, about which is a slightly raised yellowish margin which, in turn is surrounded by a reddish area. The lesions are usually multiple and they are characterized by irregular circinate borders. The term "geographic tongue" has been given to this affection because of the maplike appearance of the lesions.



Fig. 432 *Left Furrowed or Scrotal Tongue* Pain sometimes develops due to decomposing food debris retained in bottom of furrow *Right Geographic Tongue*. The tongue markings changed their position on the dorsum of the tongue from day to day

mon subjects. The lesions of geographic tongue may appear during menstruation or in association with a gastrointestinal upset. Geographic tongue recurs frequently in persons who are susceptible to this affection.

Symptoms Subjective symptoms are generally absent, but at times the patient will complain of a slight burning or irritation of the tongue when hot or highly seasoned foods are eaten. The disease is usually discovered by the patient, a friend, or his dentist. Middle-aged, apprehensive patients may develop a cankerphobia before the correct diagnosis is made.

on the dorsal surface of the tongue. An important clinical feature of these lesions is their migratory character over the surface of the tongue.

Diagnosis The diagnosis can be made in most instances by inspection. The lesions of geographic tongue may be confused with those of lichen planus. In the latter disease, the lesions have a bluish-purple color and they lack the raised yellow border with the surrounding red zone. The lesions of lichen planus are chronic in nature, and little change in their location or distribution will be noted from day to day. Tongue involvement in this disease is unusual without

wood covered with a black hairy growth similar to that which developed later on the tongue. Others consider that the bacterial forms isolated from cases of black tongue are coincidental findings rather than etiologic agents.

Symptoms. There are few if any subjective findings in black tongue. The patient or a friend will accidentally discover the abnormal coloration and papillary coating of the tongue. The elongated pa-

application of *nicotinamide compresses* to the discolored hypertrophied papillae resulted in a return to normal appearance of these structures in eight days. This form of treatment has not proven to be effective in our experience. Black tongue in humans is not analogous to canine black tongue which represents a nutritional deficiency. The application of silver nitrate has been advocated to produce a sloughing of the tongue coating.



Fig. 427: *Left:* True Black Tongue. A black, pigment producing thread form was cultured from this tongue. *Right:* Discolored Tongue. Due to the use of potassium permanganate and hydrogen peroxide as a mouth wash and medication of the gingival tissues with 8 per cent chromic acid.

pillae will at times cause tickling of the soft palate.

Diagnosis. The diagnosis is usually obvious on inspection. It is desirable to study the "hairs," the elongated hyperkeratinized filiform papillae, under the microscope and to culture several of the plucked hairs. True black tongue must be differentiated from pseudo-black tongue which results from discoloration of an abnormal lingual coating due to the use of certain medicaments, mouth washes, foods, or tobacco.

Treatment. True black tongue is not uniformly responsive to any one therapeutic measure. Schutt found that the

This form of treatment is not necessary for a benign condition like black tongue.

Since black tongue is always associated with hyperkeratinized filiform papillae, a keratolytic agent such as 10 to 15 per cent *salicylic acid* is useful in reducing the abnormal papillary growth. A *sodium bicarbonate mouth wash* should be used following the topical application of this keratolytic agent. The repeated application of 1 per cent *crystal violet* is also beneficial.

Prognosis. In spite of all forms of therapy the tongue lesions may persist for months, or they may disappear spontaneously without any treatment.

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lesions on other mucosal surfaces or on the skin

Treatment The treatment of geographic tongue is unsatisfactory. If the patient is concerned about the peculiar appearance of his tongue, his fears of cancer or other serious diseases should be allayed. The condition will disappear spontaneously in time without any form of treatment. In some patients the administration of *dilute hydrochloric acid* or *glutamic hydrochloride* seems to have a beneficial effect.

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intolerance to iodine and bromine have been ascribed as causative agents.

Pathology The pathology is characterized by edema and necrosis of the fat cells of the subcutaneous layer with relatively little increase in the interlobar



Fig. 430 Panniculitis. Sharply circumscribed area of depressed skin and result of complete loss of fat at former site of subcutaneous nodule. (Courtesy of Dr. Lovetta J. Cannon.)

connective tissue. The blood vessel walls are only rarely thickened. Fatty acid crystals are absent.

Symptoms The lesions of panniculitis range from bean size to palm size or larger. They are located more often on the thighs and occasionally on the trunk and extremities. The smaller lesions are irregular or round, bluish in

color and slightly depressed. From these ill-defined lesions morphealike plaques develop which are depressed irregular and often reach the size of 10 cm. The depth may be as much as 2 mm. in the larger plaques. The skin covering the involved areas is normal, however it presents a dark blue hue. On palpation the affected areas, during the active stage of the disease, are hard, sclerotic, freely movable irregular masses. Elevation of temperature, nausea, vomiting, and muscular pain accompany the appearance of new lesions. Recurrences may be numerous. An afebrile form characterized by the appearance of small or large indurated subcutaneous plaques occurring on the trunk has also been described. Occasionally deep-seated painful nodules which occur on the buttocks become cystic and discharge an oily or fatty yellowish or brownish fluid. The site of these healed areas is marked by a thin puckered scar. Subjective symptoms are usually absent.

Diagnosis: Recurring attacks of fever in conjunction with nodular inflammation of the subcutaneous tissue differentiate panniculitis from erythema nodosum, drug eruptions (bromide and iodide) erythema induratum, sarcoid, and paraffinoma. Whether febrile or afebrile, the final stage is a depressed area indicative of the fat loss with a pigmented, otherwise unchanged, skin covering.

Prognosis: The disease often recurs over a period of years. It may however involute and leave asymptomatic depressed scars.

Treatment The bromides and iodides are avoided. Foci of infection are eliminated. There is no specific therapy for this condition and treatment is purely symptomatic.

PANNICULITIS

SYNONYMS: *Weber-Christian disease relapsing febrile nodular nonsuppurative panniculitis, atrophy of the fatty layer of the skin, liquefying nodular panniculitis.*

Panniculitis is a rare disease characterized by irregular rounded bluish and erythematous nodules in the subcutaneous tissue

Incidence It occurs more frequently in middle aged women

Etiology The etiology of panniculitis is unknown Focal infection and



Fig. 429 Panniculitis (Weber-Christian disease) A relapsing febrile nodular nonsuppurative panniculitis.

intolerance to iodine and bromine have been ascribed as causative agents.

Pathology The pathology is characterized by edema and necrosis of the fat cells of the subcutaneous layer with relatively little increase in the interlobar

color and slightly depressed. From these ill-defined lesions morphealike plaques develop which are depressed, irregular and often reach the size of 10 cm. The depth may be as much as 2 mm in the larger plaques. The skin covering the involved areas is normal, however it presents a dark blue hue. On palpation the affected areas, during the active stage of the disease, are hard, sclerotic, freely movable irregular masses. Elevation of temperature, nausea, vomiting, and muscular pain accompany the appearance of new lesions. Recurrences may be numerous. An afebrile form characterized by the appearance of small or large indurated subcutaneous plaques occurring on the trunk has also been described. Occasionally deep-seated painful nodules which occur on the buttocks become cystic and discharge an oily or fatty yellowish or brownish fluid. The site of these healed areas is marked by a thin puckered scar. Subjective symptoms are usually absent.

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Treatment The bromides and iodides are avoided. Foci of infection are eliminated. There is no specific therapy for this condition and treatment is purely symptomatic.



Fig. 430 Panniculitis. Sharply circumscribed area of depressed skin and result of complete loss of fat at former site of subcutaneous nodule. (Courtesy of Dr. Lucetta J. Cammisa.)

connective tissue. The blood vessel walls are only rarely thickened. Fatty acid crystals are absent.

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PARAPSORIASIS

SYNONYMS: *Parakeratosis variegata*, *pityriasis lichenoides chronica*, *erythrodermia pityrialeque en plaque dissimilis*, *dermatitis psoriasiformis nodularis*, *psoriasiform and lichenoid exanthem*.

Parapsoriasis is a psoriasiform and lichenoid eruption and was first described by Jadassohn in 1804. The disease was later identified by Juliusberg as *pityriasis lichenoides chronica*. Brocq suggested the term "parapsoriasis en gouttes" in 1902 and introduced the attributes, "guttate," "en plaque," and "lichenoid" to identify three forms of parapsoriasis. However the individual cases differ markedly from one another and the clinical picture is not always clear cut.

The disease is of obscure origin and characterized by unyielding erythematous-squamous eruption free from itching and any tendency to serious complication. Parapsoriasis is exceedingly rare, occurs in young adults, persists throughout life and is very resistant to treatment.

Histopathology The essential histopathologic features of all forms of parapsoriasis consist of slight vascular dilatation, edema and leukocytic infiltration of the upper layers of the stratum corneum. Secondary changes consist of edema, moderate acanthosis and patchy epidermal parakeratosis. The granular layer may be absent. Leukocytosis is evident in the prickle-cell layer. The extensive parakeratosis and the minute dry abscesses of psoriasis are absent in parapsoriasis. Although the histologic changes in the various types of parapsoriasis are not diagnostic, histopathologic studies should be made in all cases so diagnosed clinically, since the lesions in some of the lymphoblastomata may be clinically undistinguishable from those of parapsoriasis.

Parapsoriasis en Gouttes or Parapsoriasis Guttata This is characterized

by an eruption disseminated generally over the trunk, arms, and legs. The scalp, face and hands usually escape involvement. The eruption consists of nonpruritic pea- and larger-sized pinkish or brownish red macules or maculopapules. They are oval or round, only slightly infiltrated and covered with a fine adherent whitish scale which when removed leaves a punctate purpura. In psoriasis, however, this maneuver exposes a smooth shiny surface. The eruption is persistent despite all methods of treatment and becomes more profuse as new lesions appear from time to time. The lesions seen early resemble beginning spots of psoriasis. There are no subjective symptoms.

Parapsoriasis en Plaque Parapsoriasis en plaque is characterized by rounded or irregularly shaped yellowish red patches covered with delicate thin scales. Lesions may be the size of the fingertip or as large as the hand. The sites of predilection are the trunk, thighs, and upper arms. Patches are very rarely blue or gray. Parapsoriasis en plaque has been reported to terminate as mycosis fungoides.

Parapsoriasis Lichenoides Parapsoriasis lichenoides is characterized by a yellowish red meshed network of lichenoid papules. The sites of predilection are the extremities. The trunk is very rarely involved. Lesions are often covered with fine scales. The normal integument lying between lesions enters to form the meshed network effect. Civatte described this meshed network as occurring below the normal plane of the skin and that it was really an atrophy of the skin. This meshed network is also

vated above the plane of the normal skin in parakeratosis variegata. Civatte reported cases of parapsoriasis lichenoides with outgrowths resembling mycosis fungoides.

Diagnosis Parapsoriasis is to be differentiated from psoriasis and lichen planus. The scalp and face are rarely involved in parapsoriasis. Scales of parapsoriasis are not as silvery white as those of psoriasis. Removal of parapsoriasis scales does not expose the moist and shiny areas seen in psoriasis. Lichenoid parapsoriasis never presents the flat, umbilicated papules of lichen planus. Unlike lichen planus, parapsoriasis never occurs on mucous membranes. Itching is also absent in parapsoriasis. Although parapsoriasis guttata and parapsoriasis en plaques are usually grouped together they are quite dissimilar and appear to be two different diseases, morphologically at least.

Most diseases classified as parapsoriasis are generally diagnosed clinically and by exclusion; the histologic picture is indefinite. Although many remain as the en plaque form of parapsoriasis, a fair number are clearly precursors of typical mycosis fungoides. Unfortunately in these latter cases, only time will clarify the condition.

Prognosis The character and configuration of lesions in parapsoriasis remains constant for years. Parapsoriasis is of slow progress, undergoes considerable variation, and may completely vanish. This peculiar behavior of the disease is not dependent on treatment and continues in the absence of therapy.

Treatment Treatment is of little value in parapsoriasis. Medicated baths, ultraviolet irradiation, x-ray therapy and the reducing agents employed in treating psoriasis may be of benefit in parapsoriasis.

PELLAGRA

SYNONYMS *Erythema endemium, Acute leprosy mal de la rose, Alapar scurvy mal del sol malidiano, Lombardian leprosy montazzi della miseria.*

Pellagra is a chronic constitutional disease usually occurring in individuals subsisting on an inadequate and vitamin deficient diet. The disease is not contagious; it appears endemic and runs a chronic course. It is characterized by the appearance of gastrointestinal symptoms and secondary manifestations involving the nervous system and epithelial tissues.

Pellagra was first recognized in 1735 in Spain by Casal, who observed among peasants an affection resembling scurvy but accompanied by pigmentation and roughness of cutaneous areas exposed to sunlight. Casal presented an adequate description of this disease and called it *mal de la rosa*. An identical disease,

occurring among peasants in Italy was recognized and described in 1771 by Frapolli, who named it *pellagra*.

Incidence Since 1778 pellagra has occurred in countries adjoining the Mediterranean basin including the Black Sea. Since this time the disease has been reported in all parts of the world. It is endemic in the United States, however it is more frequently seen in the southern states.

Etiology Three views have been advanced to explain the etiology of pellagra, although its origin remains obscure.

Theory of Toxic Origin The high incidence of pellagra among individuals whose diet consisted largely of maize gave rise to a toxic origin for pellagra.

It is now known that the disease occurs among individuals not subsisting on maize. Recovery from pellagra also occurs in pellagrins living exclusively on maize. This toxic theory has been discredited despite the fact that it has some supporters.

Theory of Infectious Origin. In 1910 Sambon championed an infectious origin for pellagra and contended that a pellagra infecting agent was borne by insects. He further asserted that this was probably transmitted by a species of gnat. In support of this view are the facts that pellagra occurs in an environment of neglected hygiene and poor sanitation that it occurs in persons who contacted pellagrins that there is a marked seasonal incidence and that it sometimes occurs in the well nourished.

Role of Dietary Deficiency. Goldberger presented facts refuting the infectious origin of pellagra and advanced the opinion that the disease is the result of some dietary deficiency. This view is at present accepted as the causative agent of pellagra. Roussel called attention in 1866 to the importance of diet in pellagra. Goldberger and his co-workers demonstrated for the first time in 1926 that recurrences of pellagra are prevented in susceptible persons by employing adequate dietary measures. They produced the disease in susceptible individuals by a diet previously found to produce pellagra. It was by their dietary manipulations alone that the disease was for the first time controlled and experimentally produced.

Funk in 1912 included pellagra among deficiency diseases curable by adding to the diet substances known as "vitamins."

Goldberger and Tanner designated the unknown principle as the PP or pellagra preventing factor. They included yeast and lean meat among other articles of

food rich in the pellagra preventing substance.

The pellagra preventing agent is now known as vitamin B₂ (riboflavin or vitamin G).

Pellagra is said to develop in individuals because of one of the following three causes:

1 Some individuals consume normal quantities of food but are unable to retain the pellagra preventing agent.

2 Other individuals cannot ingest normal quantities of food because they have lost the appetite for ingesting them.

3 Still another group of individuals ingest and retain normal quantities of food but for some unknown reason are unable to assimilate them properly.

The relationship of so-called "alcoholic pellagra" to endemic pellagra has long been disputed. Some investigators regard the disorder among alcoholics as a result of some toxic influence arising from spirituous liquors. This view is not accepted because alcohol deprives the addict of hunger. The fact that alcoholic pellagra is similar in clinical course to the endemic type rules out alcoholism as the causative agent. Endemic pellagra occurs among people who lack essential foods. Alcoholic pellagra on the other hand occurs in individuals who have the necessary food but who have lost the appetite for it.

Most phenomena of pellagra can be explained by vitamin B₂ deficiency. It may be also safe to conclude that pellagra is not produced by infection.

Pathology. Gastrointestinal dysfunction is the most alarming symptom of pellagra. This is characterized by enteritis and small round ulcers in the gastrointestinal mucosa. The liver and kidneys undergo fatty degeneration. The spleen undergoes atrophy and the mesenteric glands become enlarged. More than

half of pellagrins have a true achlorhydria which does not react to injections of histamine

Thickening of cutaneous areas exposed to sunlight results from infiltration of the dermis with lymphocytes. Parakeratosis of the epidermis produces roughening. The pigment in the stratum granulosum is increased.

Changes in the nervous system are

Lymphocytosis is usually present. Campbell and Shaver found that the erythrocytic mixture of pellagrins reduces iodine more rapidly than that of normal blood or of other pathological conditions. The heart is as small as in Addison's disease.

Porphyrin is usually present in large quantities in the urine. The feces contain an excess of skatol.



Fig. 481. Pellagra. Right Atrophy of tongue and stomatitis in chronic pellagra.

slight but widespread. The cerebrospinal fluid remains normal. In their order of severity of involvement, the brain is but lightly affected; the cord, especially in the cervical and lumbar regions, becomes diffusely involved, and the spinal sympathetic chains of ganglia undergo marked involvement. Microscopic changes in the nervous system include chromatolysis and degeneration of cells in the Betz layer, Clarke's column, the posterior spinal ganglia and ventral horns. Degeneration occurs in the pyramidal tracts and posterior columns.

Symptomatology. Pellagra is a chronic disease exhibiting a fixed periodicity and accompanied by gastrointestinal symptoms, cutaneous lesions, and mental deterioration. The symptoms are occasionally very acute, ending in a fulminating malignant type of the disease. The earliest symptoms include a vague languid debility, inability to concentrate, stomatitis, and gastrointestinal disturbances consisting of enteritis and diarrhea.

The cutaneous lesions are very significant. They are not common in early and

mild deficiency nor do the skin changes parallel the severity of the disease. Skin lesions are often multiform exhibiting erythema in one area, desquamation in another and pigmentation in a third. They first appear as erythematous mac



Fig. 432: Pellagra. Pigmentation following persistent or repeated attack of pellagra dermatitis, generally believed to be due to a photodynamic action on photosensitizing substances circulating in the peripheral lymphatic vessel of the skin.

ules tending to coalesce and form definite margined patches like those of sun burn. Vesicles and bullae are present in acute cases. Affected areas of the skin assume a darker hue, become rough, scaly and undergo desquamation within a few days or a fortnight. This is followed by hyperpigmentation (pellagra melanon). The diagnosis of pellagra can not be definitely established without the appearance of this characteristic disfiguring pigmentation. The early sites of cutaneous involvement are the dorsal aspects of the hands and wrists. Lesions

are distributed over the face, neck, arms, chest, and back. The feet and ankles are frequently affected. Lesions which develop spontaneously are usually symmetrical but not necessarily so. They may also develop at points of irritation, friction or pressure.

Disturbances of the gastrointestinal tract consist of dyspepsia, flatulence, and a feeling of burning discomfort in the epigastrium, vomiting and diarrhea. A very painful stomatitis and an aggravating esophagitis are often present. Speer states that the glossitis is a much more sensitive gauge than the dermatitis, both diagnostically and therapeutically. The tongue becomes flabby, large, and shows impressions of the teeth on its sides. The dorsum of the tongue becomes furred and the remaining portion undergoes



Fig. 433: Pellagra. In old case of glossitis. Probably a secondary complication since many fusospirillary organisms of Vincent were present on smear and darkfield examination.

glazing. This furring and glazing are diagnostic signs of pellagra. The tongue surface finally atrophies and becomes raw and fissured. Diffuse inflammation of the oral mucosa and the appearance of aphthous stomatitis are often seen

Sordes at the corners of the mouth may be present.

Pellagra may have a nervous onset consisting of depressed mental irritability with intermittent headache and vertigo. Deterioration of the optic nerve, melancholia, cerebrospinal symptoms and muscular tremors may occur. Other nervous symptoms include insomnia, extreme depression, inability to concentrate and coordinate happenings, poor

able in individuals in whom the disease is advanced. The mortality is not above 8 per cent, since specific therapy and adequate diet have been introduced in treatment. This compares very favorably with the mortality of 54 per cent reported a few decades ago. It is certain that exposure to sunlight is more detrimental to pellagrins than to normal people. However Smith and Ruffin state that the majority of pellagrins, under

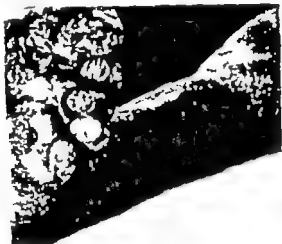


Fig. 424. Gangrene in Pellagra.

appetite with cravings for unusual things, and suicidal intent.

Diagnosis. Pellagra is not to be confused with the early manifestations of sprue which is accompanied with similar gastric symptoms and tongue lesions. Sprue is ruled out by investigating the total fats and relative fatty acids in stools, and the demonstration of megakaryocytes in the blood stream.

Beriberi does not present any difficulty in diagnosis because of the accompaniment of definite early peripheral nerve involvement and the absence of cutaneous lesions.

Prognosis. The prognosis of pellagra is favorable in mild cases and unfavor-

controlled conditions, show nothing to suggest an accentuation of the dermatitis after exposure to the sunlight.

Treatment. General Therapy. Goldberger, Wheeler and Rogers believe that the daily intake of 200 gm. (7½ ounces) of fresh meat in the form of lean beef prevents pellagra. From 2 to 4 cc (½ to 1 dram) dilute hydrochloric acid is given with and after meals. The meat is ground for patients with sore mouths. It is imperative to prescribe a basic daily diet of not less than 4000 calories. This diet is of low carbohydrate content and includes milk, lean meat, eggs, fresh vegetables, and fruits. Fresh beans and peas are especially recommended. Cane

sugar and alcohol are eliminated from the diet of pellagrins.

Favorable results have been reported from treatment by *arsenic* and *quinine* preparations.

Sabry and others reported good results from daily intravenous injections



Fig. 435. Pellagra.

of 10 cc (2¼ drams) of a 10 per cent solution of *sodium thiosulfate*. From twenty to sixty doses are necessary. *Blood transfusions* are of definite value in pellagra.

Specific Therapy. Recent studies indicate that *thiamin chloride*, *nicotinic acid* and *riboflavin* are specific in the treatment of pellagra. *Nicotinic acid* is an oxidation product of *nicotine* and is obtainable from rice polishings, yeast and rice proper. It has recently been extracted from liver and heart muscle.

Mucous-membrane lesions disappear; red cutaneous erythematous patches blanch and the other manifestations accompanying pellagra (pyralism, urethritis, vaginitis, and proctitis) are definitely improved twenty four or forty

eight hours after instituting nicotinic acid and riboflavin therapy.

Immediate sensations of warmth, tingling flushing and occasional itching follow administration of large doses of nicotinic acid. This transient reaction is more pronounced on the ears, face, and neck, and is evidenced by a rise in temperature of the skin from capillary dilation. The substitution of *nicotinamide* for nicotinic acid prevents these undesirable symptoms.

The usual daily dose for adults is 500 mg. (7¼ grains).

The daily dose for children is 100 mg. (1½ grains).

Yeast, wheat germ, hog's stomach, and liver extract are excellent adjuvants in



Fig. 436. Pellagra (Casal's necklace). (Courtesy of Dr. V. Pardo Castello.)

therapy. Yeast is the best of these adjuvants. It is indicated in cases free from diarrhea. A daily minimum of 24 gm. (6 drams) of dried powdered yeast is given. Wheat germ is an efficient sub-

stitute whenever yeast is not well tolerated. It is given in daily doses of 230 to 300 gm. (8½ to 10 ounces)

Preparations of *hog's stomach* as employed in treating Addisonian (pernicious) anemia are well tolerated by pellagrins even in the presence of diarrhea. The daily dose is from 30 to 60 gm (1 to 2 ounces) of the bacteriologically sterile dehydrated preparation.

Liver extract is given by mouth up to 100 gm. (3½ ounces) each day. Per

cent of pellagrins suffer from glossitis and stomatitis. *Dobell's solution* is highly commended as a mouth wash. Oral lesions are painted with an 8 per cent solution of zinc chloride.

DIARRHEA About 60 per cent of severe cases of pellagra manifest a diarrhea marked by frequent large, foul-smelling, liquid stools. Daily doses of tincture of opium ranging from 10 to 12 cc. (2½ to 3 minims) are given in dosage of 2 cc (½ dram). Dilute hydrochloric acid in



Fig. 437 Pellagra.

enteral therapy is especially useful. *Liver* and *hog's-stomach* extracts are selected in the regimen of treatment for their vitamin B₂ contents rather than for their antianemic factors.

The principle of *overmedication* is stressed in treating pellagra. This is accomplished by ingesting an adequate diet containing yeast or wheat germ, a sufficient dose of nicotinic acid, riboflavin and thiamin chloride, and the injection of liver extract.

Some clinicians observed improvement in pellagrins when confined in dark rooms.

Treatment of Special Symptoms in Pellagra. ORAL LESIONS. More than 50

4- to 8-cc (1 to 2-dram) doses is given each day with meals, and again three hours later.

VOMITING Vomiting occurs approximately in 66 per cent of patients with advanced pellagra. Nausea is controlled by absolute rest and use of *fluid nourishment* of 10 to 15 cc. (2½ drams to ½ ounce) of eggnog, iced peptonized milk, and ginger ale at intervals of fifteen minutes. Injections of liver extract are often beneficial. Intravenous saline and glucose may be necessary to combat dehydration.

ABDOMINAL PAIN The abdominal pain accompanying the beginning of convalescence in advanced pellagra is relieved by

sugar and alcohol are eliminated from the diet of pellagrins

Favorable results have been reported from treatment by *arsenic* and *quinine* preparations

Sabry and others reported good results from daily intravenous injections



Fig. 435: P II gra.

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Yeast, wheat germ, hog's stomach, and liver extract are excellent adjuvants in



Fig. 436 P II gra (Caudal neck) (Courtesy of Dr. V. Pardo Castello.)

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in divided doses, has been used with advantage in the macrocytic anemias associated with pellagra.

MANAGEMENT. Local medication is unnecessary because the cutaneous lesions of pellagra improve rapidly with suitable dietary measures. The skin is protected against direct sunlight, bright light, radiant heat, and irritants of all kinds.

Sponging the patient with 1:5000 solution of potassium permanganate and the

application of calamine lotion with 1 per cent ichthyol prevents secondary infection, and the latter protects the patient from the irritating effects of the sun.

The treatment of pellagra does not end by the disappearance of symptoms because of the frequent occurrence of relapses. Pellagrins are examined each month to insure continuance of a well-balanced diet rich in the antipellagra factor.

PEMPHIGUS

Pemphigus is a systemic disease of adults characterized by primary non-inflammatory bullous eruptions of cutaneous and mucous membrane surfaces of the body. The disease is not contagious. The term pemphigus formerly designated any dermatological condition accompanied by blisters, irrespective of etiology. Pemphigus is now restricted to the following conditions: (1) pemphigus chronicus or vulgaris; (2) pemphigus foliaceus; (3) pemphigus vegetans; and (4) pemphigus acutus.

Incidence and Distribution. Pemphigus is so rare that it constitutes from 1 to 2 (000 cases of skin diseases. It is equally divided between sexes. Some clinicians believe that it occurs more frequently among females in the ratio of 2:1 male. True pemphigus occurs between the ages of thirty and seventy. It is now generally accepted that pemphigus does not occur in infants. Cases of pemphigus neonatorum and pemphigus vulgaris have been reported to occur in recent years among young children of the ages of five, eight, and eleven years in the United States, England, and Germany. Whether these reported cases are identical with the disease now known as pemphigus" seems questionable.

Vocational susceptibility is said to occur among butchers and individuals contacting septic animals, especially sheep. It appears probable that other contributory factors are involved in addition to mere contact with septic animals.

Pemphigus is of universal distribution. It has been reported to occur in all parts of the tropics and Temperate Zone especially in areas where crowding, poverty and poor hygiene are conducive to malnutrition and general debility of the population. The disease is less prevalent in America than on the European continent and in the Orient.

Etiology. The etiology of pemphigus remains obscure. Conditions tending to lower the vitality and resistance of the individual are predisposing factors.

Recent evidence supports the possible presence of some etiologic factor analogous to that in deficiency diseases. This evidence is supported by known changes occurring in the blood and serous exudates of individuals suffering from pemphigus. The favorable response of individuals with pemphigus to ultraviolet light and vitamin therapy suggests vitaminosis as an etiologic factor. In 1940 two persons were inoculated with the blood

tincture of opium tincture of krameria and nicotinic acid

NERVOUS SYMPTOMS Symptoms referable to the central and peripheral nervous systems appear in 60 per cent of pellagrins. Confusion disorientation hallucination dementia and mania are the usual symptoms of central neuritis

months Normal function is restored in these neuritic areas when patients are given an adequate *high caloric diet*. Crystalline vitamin B_1 (thiamin chloride) is indicated in treating peripheral neuritis. Five to 10 mg of thiamin chloride in sterile isotonic salt solution may be given each day by the intrave-



Fig 438 Pellagra.

Peripheral neuritis is accompanied by excruciating pains requiring massive doses of *sedatives* for relief. These pains are characterized by hyperesthesia and altered reflexes about the feet and legs. The hands and arms may also become involved in hyperesthesia. Neuritis is a usual accompaniment of convalescence. The signs and symptoms of neuritis may clear up quickly or they may last for many

nous route. *Physical therapy* is often helpful.

ANEMIA More than 60 per cent of pellagrins are anemic. One third of anemic pellagrins reveal an anemia characterized by a low color index. Others have an anemia with a high color index and an increased cell volume. Most anemic patients improve by adequate *diet*. Folic acid (L. casei factor) 5 to 20 mg daily

may be accompanied by the usual symptoms of a severe systemic disturbance, namely malaise, fever, and chills.

The appearance of bullae is the first manifestation of pemphigus. Bullae oc-



Fig. 439 Pemphigus Vulgaris. Complicated by chronic nephritis.

cur upon any part of the cutaneous and mucous surfaces of the body with predilection for the face, mouth, and anogenital region. The lesions are irregular occurring singly and in groups. A bulla has a thin distended translucent wall filled with clear serum. It arises rapidly and spontaneously within a few hours or a day from an apparently sound skin without any noticeable preliminary erythema. Bullae vary in size from a pea to an egg and in number from a few to a sufficient group to cover the entire body including the scalp, palms of the hands, and soles of the feet. They occur also on the conjunctiva, in the mouth, pharynx, vagina, and within the gastrointestinal tract. Subjective symptoms in the digestive tract consist of pain on taking food. Respiration often becomes obstructed by bullae in the

nasopharynx. Cases have been reported in which the lesions of pemphigus were confined to mucous membrane surfaces, including the conjunctiva. Bullae are usually distributed bilaterally but unilateral distribution has occurred.

The duration of single lesions varies from one to two weeks. Bullae disappear without rupture by absorption and collapse of their envelopes. Sites of healing leave a dull red pigmented area which gradually disappears. Serous contents of bullae may become milky or puriform



Fig. 440 Pemphigus Vulgaris. With axillary crusting lesions. Note the bullae. Healed lesions are represented by pigmented areas. Fatal termination.

and may exhibit hemorrhage in rare cases. Excoriated bases occur about the neck, crotch, and axillae, where cutaneous surfaces meet in apposition. New bullae spring from sites of healed lesions as well as from sound skin. This

of a pemphigus patient. At no time were there untoward symptoms, and the patients have been observed recently and found to be in perfect health. This, in the author's opinion, negates the theory of a virus-borne disease.

Pathology. Bullae are the characteristic cutaneous lesions of pemphigus. They occur superficially between the stratum corneum and stratum granulosum or deeper beneath the epidermis (cuticle). There is little evidence of any inflammatory process despite the leukocytosis described by some clinicians in the upper layers of the corium. Bullae seem to result from sudden effusion of serum from the vessels in the corium following paralysis and dilation of blood vessels. Rupture of the prickle-cell layer occurs from the sudden escape of fluid into the tissue. Bullae are unilocular. Their serous contents are sterile and generally alkaline. They may become neutral or even acid in reaction. The presence of micro-organisms is generally regarded as extraneous and from secondary infection. Infection and inflammation follow rupture of bullae especially in folds of the skin where two surfaces are opposed. Excoriation occurs at these sites. Proliferation of the rete Malpighii, hypertrophy of the papillae, and inflammation of the corium occur in severe cases of pemphigus. The healing of bullae is incomplete in pemphigus foliaceus because of continued proliferation of the horny cells and the exfoliative process in which scales become incorporated in serous exudates to form crusts. The inflammatory process in pemphigus vegetans, another severe form of the disease, ends in papillomatous growths at sites of bullae.

Mucous membrane lesions show the same general characteristics as lesions of the skin. They may remain limited to

the oral mucosa for one or many months. This time lag suggests an incubation period for pemphigus vulgaris. Rupture of the mucous membranes ruptures rather quickly especially in the mouth. Mouth lesions are often accompanied by patches of superficial ulceration and erosion. Levin reported in 1920 a perforating duodenal ulcer in a severe case of pemphigus.

According to Mulvehill there is a regular consistent fall in the serum albumin fraction of blood serum proteins.

Pemphigus is free from other definite morbid anatomical findings.

Pels and Macht have shown a definite phytotoxin in the blood of pemphigus patients and in the serous exudates of lesions by conducting phytopharmacologic tests (using seedlings of the *Lupinus albus*). Their original tests revealed that the blood of eighteen patients with pemphigus possessed a constant low phytotoxic index not shown in the blood of twenty-four other dermatoses or in normal blood. Normal blood and that from other dermatoses cases gave an index of 70 to 75. The blood of the eighteen pemphigus patients showed a lower index averaging 53.8. The toxicity of the blood varied with the severity of the disease. Pels and Macht found that the serous exudates of bullae gave a lower phytotoxic index than the blood. Macht and Ostro recently reported, on 3000 suspected cases, that all dermatoses except leprosy do not give this reaction. They also noted that ultraviolet light irradiation and short exposures of filtered x radiation of the serum of pemphigus patients reduced this toxicity.

The entire skin of patients with pemphigus, according to MacCardle and his coworkers, contains increased amounts of iron, silicon, manganese, and selenium.

Symptoms. Prodromal symptoms are usually absent. The onset in acute cases



Fig. 442 *Pezophagus V. lgaris*. Note bullae and eroded areas (ruptured bullae)
(Courtesy of Dr. Carroll S. Wright.)

process may assume a chronic course with occasional remissions and exacerbations. Periods of subsidence of systemic and cutaneous symptoms occur in pemphigus, only to be followed by acute recurrences, often terminating rapidly from sheer exhaustion.



Fig. 441: Pemphigus. Note typical tense bleb just below the large lesion. These appear more suddenly on an apparently normal skin; however an erythematous halo may be present in some cases of pemphigus vulgaris.

A peculiar disagreeable odor arises from decomposing exudates and epithelial cells, such as occurs in burns and other extensive destructive skin lesions.

Nikolsky's sign occurs in every type of pemphigus, always in grave or severe types, and especially in connection with pemphigus foliaceus. It is often an early manifestation of desmolyosis or lack of cohesion between prickle cells. This test consists in determining the want of adhesion between layers of the skin in such a fashion that the upper layers of the epidermis can readily be removed by

pressure or trauma. The Nikolsky sign (a positive Nikolsky test) is the phenomenon of separation of the stratum corneum from the stratum mucosum, or even of the epidermis from the derm when the skin is moderately stroked or rubbed with the tip of the finger or a dull instrument. It may follow simple rubbing from bed linens. The Nikolsky phenomenon is also observed in bullous lichen planus and certain drug eruptions (bullous, purpuric) especially those due to sulfadiazine and phenolphthalein and in epidermolysis bullosa. It disappears as the patient improves.

Grave acute cases of pemphigus are accompanied by the usual symptoms of febrile diseases consisting of a rise in temperature (105° F) anorexia weakness, and prostration. Nervousness, insomnia and mental disturbances are often present in these cases. Subjective symptoms are mild or absent in the average case of pemphigus. When present they consist of a slight burning sensation tenderness, soreness, and itching. This itching accompanies drying and crusting and may be so intense as to make life unbearable.

Varieties. Four forms of pemphigus are identified by the severity acuteness, extent and nature of bullae. Variations in pemphigus are probably not due to different etiologic factors, but to accidental factors implanted upon the same disease. In the order of frequency and severity the four forms of pemphigus are chronic, acute, foliaceous, and vegetans.

Pemphigus Chronicus (Pemphigus Vulgaris) Pemphigus chronicus is the most prevalent and least severe form of the disease. It often runs a mild course without marked constitutional symptoms and with less cutaneous manifestations. Bullae occur in crops, with remissions, and exacerbations. The cutaneous surface is



FIG. 412 *Pemphigus Vulgaris*. Not bullae and eroded areas (ruptured bullae)
(Courtesy of Dr. Carroll S. Wright.)

occasionally free from bullae. The disease extends over a long period. Bullae tend to disappear by absorption, shrink age, drying, crusting, and shedding. Mucous membrane lesions are not common in pemphigus chronicus. Pemphigus vulgaris in old debilitated patients often develops into a severer form of the disease and ends fatally.

Pemphigus Foliaceus Pemphigus foliaceus is a rare and grave form of pemphigus characterized by imperfectly formed flaccid bullae and accompanied by generalized exfoliation. It can be engrafted upon chronic pemphigus. The disease spreads rapidly. The integument becomes moist, edematous, exfoliative and malodorous. The mucous membranes



FIG. 413. Pemphigus Vulgaris. Left: Oral and labial areas. Right: Note the large denuded or eroded areas also present on other parts of the skin.

Pemphigus Acutus. Pemphigus acutus is of acute onset, runs a limited course, and is characterized by grave constitutional symptoms. It occurs among butchers following wounds. Involved areas become denuded. The exudates are purulent and malodorous. Febrile and nervous symptoms are severe in pemphigus acutus. Many authors regard pemphigus acutus as a symptom of septicemia, while the cause of pemphigus vulgaris remains unknown.

become involved in pemphigus foliaceus. Constitutional symptoms are grave.

Pemphigus Vegetans Pemphigus vegetans is another rare and serious form of pemphigus in which luxuriant papillomas resembling condylomas develop on the excoriated surfaces left by bullae. An offensive seropurulent discharge is present. This condition is more marked about the genitalia, anus, axillae, and the inguinal folds. It may also appear about the nose and lips. Mucous membranes

lesions of the mouth and throat appear as white or red plaques. Lesions undergo crust formation and become surrounded by zones of inflammation. Grave constitutional symptoms accompany pemphigus vegetans.

Diagnosis The diagnosis of pemphigus is made by the spontaneous appear

flaccid bullae and crusted erosions, diffuse erythematous areas, especially over the nose and cheeks and upper anterior and posterior walls of the chest. The lesions often first appear in these latter areas and those on the face suggest lupus erythematosus. However bullae do not occur in lupus erythematosus.



Fig. 444: Pemphigus Vulgaris.

ance of bullae on apparently sound skin, the irregular distribution of lesions, the sequence in appearance of bullae extending over weeks or months, and the absence of constitutional symptoms in early stages of the disease. Involvement of mucous membrane surfaces of the eyes and mouth will serve to identify the disease in typical cases of pemphigus.

Senear Usher Syndrome (Pemphigus Erythematodes) This represents a variety of pemphigus vulgaris. It is apt to run a benign course for long periods of time. There are present in addition to

Pemphigus of the Mucosae Oral mucous membrane lesions develop before or after the cutaneous eruption in more than 50 per cent of patients with pemphigus vulgaris; however in those patients who develop signs of pemphigus primarily on the mucosae such signs may never appear upon the skin or only a few characteristic bullae may appear during the course of the disease. Objectively pemphigus may therefore be a disease of the mucosae only—the mouth, esophagus, eyes, nose, glans penis, or vulvae. None of these is common and at least two

are usually invaded, sooner or later at the same time (conjunctivae and oral mucosae)

When it appears and remains limited to the conjunctivae (it may begin in one

disease; they do not occur later in the disease. For other signs incident to this condition the reader is referred to text books on ophthalmology. Ocular lesions suggesting those mentioned above have



FIG. 445 Pemphigus Vulgaris. Early (ten weeks duration). Flaccid bullae crust-covered erosions, potholes. Noticeably signs. Additional lesions developed later over the entire body with fatal termination.

or both) it is termed ocular pemphigus or eventual shrinkage of the conjunctiva. The latter term is descriptive of the final stages of the disease. Transitory vesicles or bullae are observed on the cornea or conjunctivae from time to time, early in, and during, the course of the

been observed in conjunction with the cutaneous lesions of dermatitis herpetiformis and epidermolysis bullosa. Involvement of other mucosal surfaces, such as the mouth, may precede, accompany or appear subsequent to involvement of the conjunctivae. The character

Pemphigus

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phases of Darier disease are similar to it, however. Fractional unfiltered x-ray radiation appears beneficial.

Differential Diagnosis: Pemphigus must be differentiated from urticaria (urticaria bullosa), erythema multiforme, dermatitis herpetiformis, dermatitis venenata, impetigo, syphilis, epidermolysis

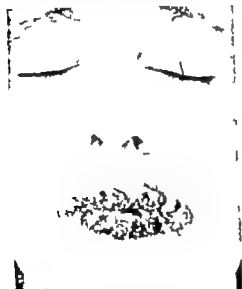


FIG. 447 Pemphigus Vegetans. Of 15 to 20 years duration. Groin and umbilical lesions were also present.

bullosa and the bullous eruptions accompany intolerance to drugs (luminal, phenolphthalein, chloral hydrate, bromides, and iodides). Pemphigus is identified by the characteristic bullous eruptions. Clinically typical dermatitis herpetiformis has been seen to eventuate in typical fatal pemphigus vulgaris.

Urticaria is characterized by wheals, dermatographia, and a history of the disease. The lesions of *dermatitis herpetiformis* vary in form, have a tendency to circinate grouping and are accompanied by severe itching. *Impetigo* reveals blebs containing pus, is infectious,

and automuculable. *Epidermolysis bullosa* is congenital. It follows trauma. Bullosa *syphilitica* occur in early congenital syphilis. It is ruled out by the age of the patient, a positive Wassermann reaction, and its response to anti-luetic treatment. *Dermatitis venenata* is ruled out by its history and the course and character of the lesions.

Prognosis The prognosis of pemphigus is usually unfavorable especially in severe forms of the disease. Recovery usually takes place in pemphigus chronicus. The absence of constitutional symptoms, the presence of a small number of bullae and the tendency of lesions to heal are favorable indications in prognosis. Pemphigus chronicus can change by passing into one of the more severe form of the disease.

Pemphigus acutus is usually fatal. The extent and character of lesions, together with the severity of constitutional symptoms, are indicative of the outcome. Milder cases recover but more severe cases end fatally.

Pemphigus foliaceus may continue over a longer period. Death sets in from intercurrent conditions like pneumonia and uremia.

Pemphigus vegetans may continue for a few months or a year but ends fatally.

The degree of mucous-membrane involvement is an index to the severity of pemphigus. The difficulty of maintaining nutrition is always a serious handicap in treatment.

Recovery has been reported in all forms of mild pemphigus. The prognosis is, however, decidedly grave in cases of severe pemphigus.

Treatment The treatment of pemphigus is largely supportive and palliative because of want of specific therapy. Adequate hygiene and general supportive measures consisting of a nourishing

istic lesions are vesicles or bullae. Because of the heat, moisture and trauma they rupture early leaving oval or round red erosions of varying size which may become confluent to form larger irregular eroded areas. The usual sites in the mouth are buccal, labial and gingival mucosa.

Eventually the affected mucosal surfaces assume a smooth scleroatrophic ap-

Familial Benign Chronic Pemphigus (Hailey and Hailey) At least five different, mostly histologic, designations have been given to this condition: dyskeratoid dermatosis, keratosis follicularis with vesiculation, recurrent herpetiform dermatitis, and dyskeratosis bullosa hereditaria.

The disease occurs in more than one member of the family and the sites of



Fig. 416 Pemphigus. Note the tense (though they may be flaccid) bullae below the left labial angle; the white areas represent the sites of healed bullae.

pearance with erosions here and there to indicate the character of the primary lesion. In addition there is a general tendency to atresia of the involved cavity. Bullae indicating the nature of the oral, conjunctival, pharyngeal or vaginal disturbances, may appear at any time during the course of the mucosal involvement. Pemphigus, limited to the mucosae, is benign as to life, malignant as to local disturbances; it is persistent, chronic, resistant to therapy, and progressive.

predilection are the collar area of the neck, the axillae, around the waist line, and the groin. The lesions consist of patches of vesico-blebs accompanied by secondary erosions and crusts. Active lesions are found at the periphery of these patches. There is usually a positive Nikolsky test. The diagnosis depends largely on the histologic finding of dyskeratotic cells in the walls of the bullae, thus differentiating it from pemphigus clinically and histologically. Certain

ganisms. Bone acid dusting powder 1 per cent aqueous solution of aluminum acetate Bennett's solution ($\frac{1}{2}$ dram sodium carbonate in 1 pint of distilled water) or linimentum calcis is recommended for topical application.

Immersion of patients in a bathtub of caron oil with 10 per cent ichthylol at

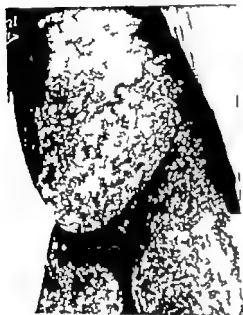


Fig. 449 Pemphigus. Granulomatous lesions seen in pemphigus vegetans.

blood temperature an hour each day for several successive days will give great relief when large areas of the body are covered with flaking adherent crusts. The bath with caron oil and ichthylol is soothing and comforting. It serves to detach crusts and cleanses surfaces difficult to treat effectively by other methods.

A solution of 2.5 per cent carbolic acid has been recommended as local antiseptic dressings in cases of pemphigus vegetans.

Dressings which produce irritation and are difficult to apply and remove are avoided. However pressure bandages

(as outlined under Burns, see p 261) and applied to extremities and trunk are definitely beneficial in pemphigus foliaceus (Riley). They have been used in pemphigus vulgaris with benefit (Quequerre). Theoretically they should help prevent excess loss of serum protein and possibly retain any antitoxic or antibacterial factors present in such serum. When used odors from decomposing desquamation tend to disappear. The following should be used on the skin between it and the pressure bandages. Spray entire skin surface with a 1:1000 solution of phenol and follow this with applications of cholesterolized petrolatum (aquaphor).

Mucous-membrane surfaces are kept clean by irrigation with flavored sodium perborate neutralized by monocalcium phosphate.

Arsenic, iron, quinine, and strychnine have been widely used in pemphigus. Arsenic in the form of Fowler's solution or the Asiatic pill (arsenic trioxide with pepper) given to the degree of tolerance, is a most popular internal medication. Subcutaneous injection of 2 per cent aqueous solution of sodium arsenate is perhaps the best method for administering arsenic. Pemphigus patients are sometimes very tolerant to arsenic. The limit of tolerance is anywhere from 80 to 100 minims of a 2 per cent solution. Stovaine or acetarsone has been successfully employed in Vienna. Oppenheim recommends one tablet, 0.25 gm. (375 grains) of acetarsone daily for three days with a full glass of water one-half hour before breakfast, then a rest period of three days. During the next three days, give two tablets daily, allow another three-day rest period, and then give a dosage of two, three, and three for three days, etc. This is continued until patient has received as many tablets as he weighs in kilograms. This drug is

diet rich in vitamins, are important. Rectal alimentation is instituted when ever painful bullae in the mouth render feeding difficult.

The *continuous bath* is the most useful of all local measures. The temperature

luous bath an ideal method of treating skin lesions in which large areas of the body are denuded. The prohibitive cost of this regimen of treatment limits its universal adoption in all cases of pemphigus.



FIG. 418: Pemphigus Vegetans. Scalp, neck, and groin were likewise similarly involved. Three years duration.

and salinity of the bath should be the same as the blood. This method of treatment affords the greatest relief to patients suffering from extensive, severe, exudative skin lesions. Cleanliness, the avoidance of dressings, general support of the body with minimum pressure, and protection from air make the con

In the absence of the continuous bath the contents of bullae are evacuated and denuded areas are disinfected by sprays, powders, or ointments. Sprays and wet dressings of potassium permanganate or 1 per cent gentian violet (or 1:1000 aqueous solution) will prevent secondary infection with pyogenic or

ganisms. Boric acid dusting powder 1 per cent aqueous solution of aluminum acetate Bennett's solution ($\frac{1}{2}$ dram sodium carbonate in 1 pint of distilled water) or *linimentum calcei* is recommended for topical application.

Immersion of patients in a bathtub of caron oil with 10 per cent ichthol at

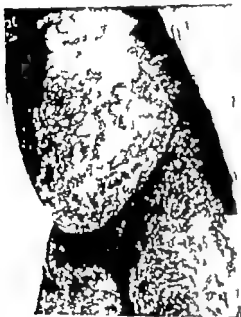


Fig. 449 Pemphigus. Crustulomerosed lesions seen in pemphigus erythem.

(as outlined under Burns, see p 261) and applied to extremities and trunk are definitely beneficial in pemphigus foliaceus (Riley). They have been used in pemphigus vulgaris with benefit (Quequerre). Theoretically they should help prevent excess loss of serum protein and possibly retain any antitoxic or antibacterial factors present in such serum. When used, odors from decomposing desquamation tend to disappear. The following should be used on the skin between it and the pressure bandages: Spray entire skin surface with a 1:1000 solution of phenol and follow this with applications of *holsterolized petrolatum* (aquaphor).

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blood temperature an hour each day for several successive days will give great relief when large areas of the body are covered with foul adherent crusts. The bath with caron oil and ichthol is soothing and comforting. It serves to detach crusts and cleanses surfaces difficult to treat effectively by other methods.

A solution of 2.5 per cent carbolic acid has been recommended as local a septic dressings in cases of pemphigus erythem.

Dressings which produce irritation and are difficult to apply and remove are voided. However pressure bandages

especially valuable in pemphigus vegetans and in benign or early cases of pemphigus vulgaris. It may be continued until signs of intolerance develop. The Davis treatment consists of intravenous injections of iron cacodylate or sodium

injections of horse serum or foreign proteins have been recommended by many clinicians. Thromboplastin is about the most serviceable foreign protein. Auto-serotherapy and blood transfusions are beneficial. Intravenous gl



Fig. 450 Pemphigus Vegetans. Scalp, axillae and neck were similarly involved. Three years duration.

arsenate with subcutaneous injections of coagulin (Ciba). Grindon modified the Davis treatment by substituting tryparamide for sodium arsenate. He selected tryparamide because of its low toxicity and value in raising tissue resistance. Hypodermic injections of 1.5 gm (22 grains) of coagulin are given on alternate days in conjunction with tryparamide

case therapy is useful because of its protective effect on the liver. Good results have been obtained by alkalinizing the body fluids with sodium bicarbonate, the citrates, and the citrocarbonates.

Salicin up to 2 gm (30 grains) in water three times each day has been advised. Pilocarpine and atropine are indicated in some cases. Prolonged ad

administration of *thyroid extract* without reaching maximum tolerance has been recommended by Torney.

Pemphigus is, in the light of our present knowledge, a syndrome complex of some deficiency rather than a specific



Fig. 451 Pemphigus Vegetans. Both palms, axillae, arms, and both axillae were involved. Note the isolated lesions. Remains of bullae could be observed at the margins of most of these.

disease. Its inclusion among deficiency diseases is justified.

Clinicians agree that *ultraviolet light* produces beneficial results in pemphigus patients. It is an established fact that the irradiation of foods and living animals by ultraviolet light brings about the factor supplied by vitamin D. The importance of a diet rich in vitamins is stressed by all clinicians. Viosterol is given in daily doses of 100,000 to 500,000 units for five days of the week. Nausea, diarrhea, epigastric pain, and

cardiac disturbance (characterized by arrhythmia) may follow high viosterol administration. It is then discontinued until symptoms have disappeared.

Macht and Ostro advise *x radiation* over the liver and spleen.

Germanin (naphunde) (Bayer) is a complex urea compound employed with usefulness in early cases of pemphigus. Intravenous and occasional intramuscular injections of 0.15 to 1 gm. ($2\frac{1}{2}$ to 15 grains) of germanin in a series of from four to ten treatments at intervals of two or three days have been advised.

Snake venom, *sulfapyridine*, *liver extract*, *vitamin C*, *nicotinic acid* and *riboflavin* are other preparations employed for treatment of pemphigus. Each of these preparations has its advocates.

Supportive and symptomatic treatments as employed in grave systemic diseases are essential for pemphigus.

An easily assimilable, nutritive diet rich in vitamins B and D is urged.

The author recommends increasing doses of viosterol starting with 80,000 units daily for five days a week. This medication is stopped when symptoms of cardiac distress supervene. Daily doses of arsenate of copper are given to the point of arsenical tolerance. General *ultraviolet-light therapy* is also given daily. Whenever possible blood transfusion from a cured pemphigus patient is given. *Liver* is given parenterally if anemia occurs. Recurrences are usual at intervals during a period of two years succeeding the primary attack. No favorable results have been seen by the author following the use of the sulfa drugs, including sulfapyridine which has given good results in the treatment of dermatitis herpetiformis.

PHAGEDENA

The term "phagedena" is used to indicate the marked tendency of certain ulcers to invade surrounding tissues rapidly.

Etiology Phagedena develops in ulcers arising from various causes. It deserves clinical recognition because it results in a condition different from that in which it has commenced. General belief is that it represents a superadded infection by organisms of special virulence or by several organisms acting in symbiosis. Phagedena occurs in the robust as well as in the debilitated. The commonest organisms found as indicated by a study of the literature on progressive ulceration are generally aerobic and anaerobic hemolytic and nonhemolytic staphylococci and streptococci alone or together in various combinations.

Symptoms The onset is more or less sudden with the original ulcer assuming a rapid or fitful progress. The ulcers are acute in their general appearance chronic in their duration. Their chief characteristics—destructive appearance, prolonged evolution, and acute local activity—differentiate them from the occasionally seen extensive ulcer due entirely to glanders, cancer, granuloma inguinale, tuberculosis, and malignant secondary or tertiary syphilis, in which however phagedena may develop.

In phagedena the destruction may be slight or in due time very extensive, the extent depending on the duration and on the degree of activity. Ulceration may tend to heal at one point, only to progress at another.

In several weeks or months larger areas may be destroyed. The skin of the entire abdominal wall, or on the face, the nose, lip, and cheek—on the genitalia, the vulva, thighs and buttocks, or the penis, scrotum, and thighs. The entire skin

thickness is involved and in the deeper types the underlying muscles, tendons and vessels may be exposed. The ulcer itself is irregular often polycyclic; the perpendicular inclined or undulating borders are marked by a red infiltrated zone; the coating of thick yellowish pus and cutaneous debris when removed exposes a red granulating irregular surface with crevasses here and there. The draining lymph nodes are often enlarged and painful (Darier).

Phagedena in Chaneroid

This type of phagedena occurs in the groin or on the genitalia in a primary chaneroid or in a chaneroid developing



Fig. 452: Phagedenic Chaneroid. Post-tetanic inoculation on right arm.

as a result of a broken-down inguinal bubo. It develops several days after the original condition has presented itself. It is the commonest type of phagedena. The ulcer enlarges rapidly as a result of the formation and confluence of dermic and subdermic pus collections, and it has the tendency to advance upon the surface tissues rather than to extend in depth.

Phagedena in Chancre

This is exceedingly rare and must not be confused with giant chancres or mixed chancre—chancroidal lesions.

Phagedena in Ulcerative Syphilides

Occasionally phagedena develops in a tertiary lesion. There are apparently

lating in persons who appear to maintain their general health for many months to several years. In these however the important etiologic factor in most cases appears to be syphilis, since rapid improvement ordinarily follows *antisyphilitic therapy* in others, *shock therapy* such as typhoid vaccine intravenously is a necessary addition to the ordinary therapy

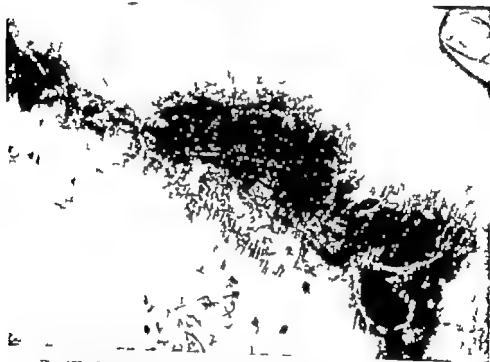


Fig. 483 Phagedena Geometrica. Phagedenic ulcer region, right lower abdomen, followed incision of an inguinal abscess three years previously. Note thinning, undermined borders of the ulcers and the extensive scarring of preexisting, but healed, lesion at site of original incision. Healing of the ulcer at one point with progression at another is evident and was observed in this patient.

predisposing factors, such as anemia, debility or diabetes. The center of the face and the genitalia are the commonest locations. The destruction may be severe enough to involve not only the nose, upper lip, and cheeks, but the underlying bones and palate as well. The process is slowly but progressively multi-

Phagedena in Pyoderma

SYNONYMS *Phagedena geometrica* (Brocq) *pyoderma gangrenosum* (Brunsting) *chronic undermining burrowing ulcer* (Meleney) *postoperative suppurative progressive ulceration*.

Symptoms This condition is in all probability both primarily and second

PHAGEDENA

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Symptoms The onset is more or less sudden with the original ulcer assuming a rapid or fitful progress. The ulcers are acute in their general appearance, chronic in their duration. Their chief characteristics—destructive appearance, prolonged evolution, and acute local activity—differentiate them from the occasionally seen extensive ulcer due entirely to glanders, cancer, granuloma inguinale, tuberculosis, and malignant secondary or tertiary syphilis, in which however phagedena may develop.

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In several weeks or months larger areas may be destroyed—the skin of the entire abdominal wall, or on the face, the nose, lip, and cheek—on the genitalia, the vulva, thighs and buttocks, or the penis, scrotum and thighs. The entire skin

thickness is involved and in the deeper types the underlying muscles, tendons and vessels may be exposed. The ulcer itself is irregular often polycyclic, the perpendicular inclined or undermined borders so marked by a red infiltrated zone; the coating of thick yellowish pus and cutaneous debris when removed exposes a red granulating iridescent surface with crevasses here and there. The draining lymph nodes are often enlarged and painful (Darier).

Phagedena in Chancreoid

This type of phagedena occurs in the groin or on the genitalia in a primary chancreoid or in a chancreoid developing



FIG. 452: Phagedenic Chancreoid. Following autoinoculation on right arm.

as a result of a broken-down inguinal bubo. It develops several days after the original condition has presented itself. It is the commonest type of phagedena. The ulcer enlarges rapidly as a result of the formation and confluence of dermic and subdermic pus collections, and it has the tendency to advance upon the surface tissues rather than to extend in depth.

The more extensive the lesion, the more evident are these characteristics. It is possible to auto inoculate with the pus.

Treatment Treated early phagedenic lesions are easily cured, great attention must be paid, however to detailed antiseptics of every crevice, and



Fig. 456 Phagedena Gangrenosa. Showing healed and several large active lesions.

especially those underlying the borders. The *sulfonamides* orally administered are occasionally of value. In extensive cases, *penicillin* may be tried. All types are benefited by local therapy preferably with *activated zinc peroxide* or a 15 per cent suspension of finely powdered *sulfathiazole* in aqueous solution of *phenol* (1:1000). If *zinc peroxide* is to be used, one should cleanse the surface of the ulcer with *boric acid* solution and then pack the powder

thoroughly into all crevices and over the floor of the ulcer. This treatment should be given daily. Pus gradually disappears as the base and borders granulate towards cicatrization. If the *sulfathiazole* suspension is to be used, one should allow it to stand and with a sterile applicator take up as much of the powder as possible from the bottom of the bottle and use this as packing for each crevice. The results are equally good.

Phagedenic Tropical Ulcer

STROVYN Tropical Ulcer

These ulcers, common in tropical and subtropical countries, are generally situated on the legs and dorsa of the feet. Following an abrasion or cutaneous break from any cause, such as a scratch or insect bite a pustule develops which is early converted into a rapidly spreading ulcer. The ulcer advances both on the surface and in depth, often involving tendons, nerves, joints and bone. The fusiform bacillus and spirillum of Vincent have been found most often in tropical ulcers, but these ulcers are thought to have a varied causation, and the rôle played by Vincent's organisms has yet to be determined. It is also believed that many of these ulcers have such basic causative factors as syphilis, chancreoid, leishmaniasis, leprosy, yaws, ground-itch (uncinariasis) and ulcers of sickle-cell anemia.

Treatment Therapeutic measures are directed at both the basic lesion and the local infection (see Ecthyma, p. 318). Gill found *sulfanilamide* powder locally satisfactory. Cohen and Pfaff state that *penicillin ointment* (calcium penicillin, 100,000 units, in equal parts of lanolin and cold cream) freshly prepared every forty-eight hours is specific.

arily of simple pyodermatous origin with organisms of a special virulence acting alone and in symbiosis. The process begins in (1) an ecchymiform lesion (2) in normal skin injured by surgical or spontaneous evacuation of a

like that already described for phagedena. However Brocq under the term "phagedena geometrica," has described a special objective form characterized by (1) sharply defined borders with the lesions appearing in circles or parts of



Fig. 454: Phagedena Geometrica.



Fig 455 Phagedena Geometrica. *Left* Superimposed upon hidradenitis suppurativa. *Right* Not regularity of the undermined, preading borders.

subcutaneous (abdominal axillary or inguinal) pus collection and (3) in post operative wounds. It is seen in those debilitated by such diseases as ulcerative colitis and pulmonary tuberculosis, but may occur in those in apparent good health. The clinical appearance is

these and (2) by the fact that the periphally extending ulcerative process involves not only the skin but often its entire thickness. The borders are undermined and swollen with small purulent cavities and infiltrated skin beneath the erythematous halo surrounding the ulcer

Abnormal Pigmentation Abnormal pigmentation may be produced in one of the following ways.

1 By deposition of hemoglobin and its derivatives, as seen in the purpuras and ecchymoses. Localized, especially to the

stances as silver and bismuth. It may also follow the intake of arsenic, the local use of pigments, as in tattooing, and the use of cosmetics containing aniline dyes or metallic pigments, such as lead, mercury and bismuth. In these latter it is not known if the slate-colored staining is due to the metal or to an increase in melanin. Both organic (trivalent and pentavalent) and inorganic arsenical (Fowler's solution, sodium cacodylate) may cause with or without a preceding



Fig. 457 Pigmentation. Bismuth line at gingivodental margin, with labial mouth pigmentation of the labial mucosa. (Courtesy of Dr. Jacques P. Gueguierre.)



Fig. 458 Pigmentation. Perioral, due to phenolphthalein. This pigmentation must be differentiated from *hemorrhagic eruption* (Pott) which is perioral, sometimes facial, brownish-black pigmentation occasionally seen in young girls at the first menstrual period. It must be differentiated from *Brocq's perioral* pigmentation erythroid which is brownish-red, varies in intensity from day to day and largely disappears under vitropression.

legs, is the more or less permanent pigmentation largely due to this pigment as seen in hemostatic dermatitis and in progressive pigmentary dermatoses of Schamberg. In Schamberg's disease there are irregularly sized and shaped, brownish patches in which, or at the borders, are the typical erythematous cayenne-pepper like puncta. The lesions are located higher up on the leg than those seen in hemostatic dermatitis.

2. As direct abnormal production of melanin, as in mongolian spot, pigmented naevus, neurocarcinoma, and melanotic sarcoma.

3 By deposit in the skin of such sub-

arsenical dermatitis, an increase in the melanin of the skin (the pigmentation is not due to the deposition of arsenic in the skin). It is a diffuse, spotty or speckled pigmentation usually beginning

Veldt Sore

SYNONYMS: *Desert sore* *Barcoo-Rot*.

This is a form of tropical ulcer in which a variety of organisms—strepto-

cocci staphylococci, and especially the Klebs-Loeffler bacillus—has been found. Diphtheritic palsies have been observed in some patients (see Cutaneous Diphtheria p 245)

PIGMENTATION

The color of the skin varies in different races and in different parts of the skin of an individual

Pigmentation due to melanin is normally marked in the axillae, areolae of the nipples, external genitalia and perianal region. It is also true that pigmentation due to melanin shows a physiological increase in females during pregnancy. White skin grafted into a dark person gradually acquires a dark color. Black skin grafted into a white person gradually loses its color. These observations indicate that the epithelium does not directly form melanin and that this pigment may arise from the connective tissue and the wandering cells. Besides the pigment melanin the skin may in certain conditions be tinted by carotene and by hemosiderin or reduced hemoglobin.

Formation of Pigment Melanin
There are three types of pigment forming or pigment-containing cells, in the skin: the cells of the stratum germinativum, the dendritic cells (cells of Langerhans) and the wandering phagocytes (perivascular histiocytes) of the corium (also called chromatophores). The hemosiderin in hemosiderosis is also found in these histiocytes.

Bloch is of the opinion that the cells of the epidermis are the only cells able to form pigment and that each cell of the stratum germinativum is a potential melanoblast. He also asserts that, under certain circumstances, the mesoderm is capable of producing pigment forming cells. He cites, as an example, the mela-

nin of the "mongolian blue spot," which is of mesodermic origin.

Kyrle believes that two factors are necessary for pigment formation: (1) a mother substance belonging to the phenol group called "tyrosin," and (2) a specific ferment which oxidizes this colored substance and produces a colored end product known as "melanin."

Bloch applied this phenomenon to man by concluding: (1) The skin of man must possess an oxidizing ferment with a specific action on dioxyphenylalanine (an amino acid) or dopa. This ferment is present whenever there is melanin. Bloch calls this ferment "dopa-oxidase" or "dopase." (2) The human pigment "melanin" is a product of a mother substance whose chemical structure identical with dioxyphenylalanine (dopa).

The influence of light in the development of pigment in the forms of bronzing and freckles in the white race is well known. Not all whites develop pigment in response to light stimulation, perhaps owing to the fact that some skins are more fluorescent than others. Melanin formation is influenced by mechanical, physical (other than sun-light) and chemical agents, and these latter by photosensitization such as the acridin compounds including atabrine by the endocrine secretions (arsenicals, pituitary and gonads) and by certain vitamins (A, B and C). Pigment is absent in albinos, present less often in blonds than in brunets, and found most frequently in the dark skin, racial group-

storage disease, Gaucher's primary idiopathic splenomegaly, Niemann-Pick's disease, pituitary disease, ochronosis, leprosy, hepatic disease, pancreatic disease, diabetes mellitus, lymphoblastoma (Hodgkin's disease), lymphosar-

comas of adiposogenital dystrophy.

In ochronosis, there is alkaptonuria, and the pigment in local or diffuse areas is deposited in the skin, cartilage, and eyes. The ears and nose may be bluish. Skiagraphic study especially of the intervertebral discs, and slit-lamp study of the eyes are indicated. The pigment in the cartilage of the ears prevents transillumination. In hemochromatosis (the triad of hepatic cirrhosis, diabetes mellitus, and pigmentation) the pigments are hemosiderin and melanin. The condition is a rare chronic affection usually seen in males past middle life. Asthenia



Fig. 461 Pigmentation. Due to chronic malaria.

coma, all forms of leukemia, atrophic arthritis, xeroderma pigmentosum, and urticaria pigmentosa. In these the cause may be complicated by arsenic and x-ray therapy. The pigmentation in Addison's disease is diffuse but more marked in the normally pigmented areas and those parts exposed to pressure and sunlight. It first appears on the dorsa of the hands, especially the knuckles; the face and neck, axillae, groin, perineum, anus, and genitalia are favorite sites for its occurrence. The oral mucosa may be involved. Pigmentation in circumscribed or diffuse forms has been observed in such pituitary diseases as acromegaly, pituitary basophilism (Cushing's syndrome), pituitary cachexia (Simmonds's disease), pituitary dwarfism, and the several va-



Fig. 462 Pigmentation. Of skin due to vitamin-A deficiency. Anterolateral aspect of right knee showing the intense pigmentation and the dark follicular keratosis (phrynoderma). (Courtesy of Dr. Jesse A. Tulaczek.)

is usual but diabetes may be absent. The iron pigment, hemosiderin, may be demonstrated in the skin by the Fishback test. A few drops of equal parts of sterile 0.5 per cent potassium ferrocyanide and 1:100 normal hydrochloric acid are injected intradermally. The skin

on the trunk. The oral mucosa may be involved. Arsenical palmar and plantar keratoses are usually present. In argyria (see p 88) which may result from the prolonged use of a silver compound (e.g. argyrol eye or nose drops) there is a per-



Fig. 459: Pigmentation. (In Simmonds' disease [hypophyseal ca- cectasia]) Note loss of pubic hair and generalized pigmentation

manent pigmentation due to reduced silver deposition. The color is bluish and in many instances is mistaken for cyanosis. The pigmentation is usually generalized but may be localized to the conjunctiva in particular. Certain chemicals acting by absorption or directly on the skin, turn it yellow (tetra-yl used in explosives, nitric acid and picric acid). Others, after ingestion cause the same color; (atrabrine, trinitrotoluene, trypt- flavine and dinitrophenol) with or with-

out liver damage and accompanying icterus. The presence of atabrine may be detected by examining the lunulae of the nails with Wood's light: there is fluorescence.

4 By the ingestion in excess of certain foods, such as carrots, spinach, pumpkin, squash, paw paws, peaches, apricots, tomatoes, green peas, oranges, red beets, yolk of egg producing carotenemia (xanthemia) and a yellowish skin pigmentation (carotinoderma). The yellow pigment, carotene, which is normally present in the skin and subcutaneous fat, is in this latter state, especially exaggerated on the palms and soles and nasolabial furrows. Carotenemia has been observed also in eunuchs, myxedema, and Simmonds' disease. The sclera are not stained as they are in jaundice, and the icterus index is normal. However scratch-



Fig. 460: Pigmentation. Buccal and labial mucous-membrane pigmentation in Addison's disease

ing because of the pruritus, may lead to an increase in the melanin also.

5 As the result of organic disease, as in Addison's disease, uterine disease, exophthalmic goiter, urticaria, cancer, bronze diabetes (hemochromatosis), chronic malaria, Von Gierke's glycogen

■ A peculiar artificial discoloration of the skin may follow employment of eau de cologne and toilet preparations containing ethereal oil (oil of bergamot, oil of lemon, and oil of lavender). This type of pigmentation is known as berlock dermatitis. According to Pierini, peribuccal pigmentary erythrosis (Brocq) is a circumscribed form of Riehl's melanosis due to the photodynamic action of tinted face powders. In this melanosis, there are three chief features: selective occurrence in females, clear brown color and predilection for perioral or central areas of the face.

Chloasma

Chloasma identifies pigmentary affections of the skin arising as idiopathic and symptomatic signs of internal disease (see also p. 219).

Chloasma Uterinum Chloasma uterinum is a peculiar yellowish brown pigmentation, occurring chiefly about the face during pregnancy in uterine and ovarian disease, and occasionally in connection with peritoneal tuberculoles and carcinoma of the abdominal viscera. It disappears on the return of menses when it is due to pregnancy. It also disappears when due to systemic or organic disease on removal of the causative agent. Cornbleet has advised large doses of *Vitamin C* in persistent cases.

Chloasma Bronzium (Tropical Mask) Cantlie called attention to the occurrence of chloasma bronzium among Europeans living in tropical and torrid climes. The face and chest of Europeans inhabiting these areas gradually become pigmented and assume the appearance of a bronze mask. The mucous membranes are not affected. The condition disappears gradually on return to Europe.

The treatment of chloasma bronzium consists of employing peelings and bleaching preparations which contain salicylic

acid or bichloride of mercury but they are not free from harm.

The following formulas are among those used for bleaching

I

Hydraz. ammoniat	2.00
Hiemath glycoler	2.00
Ung. aquaphor	30.00
Ol. limonis	0.12

Use. Apply once daily

II

Hydraz. chlor. corros.	0.25
Acid. hydrochlor. dilute	4.00
Glycerin	30.00
Alcohol	30.00
Aq. roseae	300.00

Use. Apply twice daily

III

Concentrated hydr. peroxide	6.00
Aquaphor	30.00

Use. Precede the application of this cream by application of ammoniac water (diluted 1:20) and allowed to dry on the skin.

Industrial Melanoderma

A case of industrial melanoderma, reported by Schestokowa, consisted of an eruption of blue-violet macules on the cheeks and forehead of a telephone operator six weeks following a severe electric shock. The condition disappeared in the course of two months.

Scarpa called attention to tar melanoses. He reported three cases of brown-violaceous discoloration of the skin, with a reticular distribution over uncovered parts of the body in patients working in an atmosphere laden with the vapors of some fossil tar. He championed the necessity of instituting prophylactic measures in the tar trades. Both pitch and tar melanoses are the result of photosensitization resulting from exposure of pitch or tar-stained skins to specific spectral bands of light (Foerster and Schwartz). Riehl's melanosis is a pigmentation of the face, neck, and forearms due to the photodynamic action of tar, tar derivatives,

gives a blue to purple wheal at once in the presence of hemosiderin. A biopsy will also reveal the iron deposition. In uremia the skin may be yellowish, presumably the result of a retention of the urinary chromes. The skin is stained yellowish to green in icterus due to reten-



Fig. 463: Pigmentation. Postpellagral, dorsa of hands.

tion of bile pigments. The normal icterus index is 4 to 0. Subclinical icterus is from 0 to 15, but above this the conjunctiva under a good light will have a yellowish tinge. The wheal produced by the injection of histamine will be yellowish if bilirubin enters it, be yellower than the surrounding skin.

0. Inanition may alter the color of the skin. This is frequently observed in conjunction with gastrointestinal diseases, such as celiac disease, tuberculosis of the ileum, gastrojejunal fistulae or intestinal polyposis. The skin assumes a pale yellow color and the condition is referred

to as "chlorasma cachecticum." Inanition may be accompanied by vitamin deficiencies. Vitamin A deficiency has been associated with a varying degree of pigmentation of the skin and conjunctivae. The pigmentation is brownish to slate and has been observed to disappear after the administration of cod-liver oil and the correction of this deficiency. Vitamin C also plays a part in melanin production. This vitamin appears necessary for the complete metabolism of tyrosin and phenylalanine. In any case, pigmentation of the skin is common in scurvy and disappears following correction of this vitamin deficiency. Vitamin C therapy tends to decrease the hyperpigmentation of Addison's disease. Pigmentation is also found in pellagra (see p. 581) and in sprue. In the anemias in general, the skin often has a lemon-yellow color and occasionally a diffuse pigmentation is observed.

7. As the result of overnutrition, especially when food rich in lipochromes is ingested. This may produce a yellowish discoloration of skin (carotinoderma).

8. As the result of certain inflammatory skin diseases, especially following sunburn, erythema ab igne, lichen planus, psoriasis, syphilis, dermatitis herpetiformis, pemphigus, exfoliative dermatitis, and eczema. It is observed also in such skin diseases as neurofibromatosis, pigmented urticaria, scleroderma, acanthosis nigricans, and parasitic melanoderma. In this latter (chronic infestation with pediculosis corporis) seen in tramps and vagabonds, other factors may be present to account for the pigmentation (aside from constant scratching). These factors may be chronic alcoholism and the resultant avitaminosis. The pigmentation is especially marked over the nape of the neck, the upper back and shoulders, and the waistline.



Fig. 464 Pinta. A Erythemasquamous form. B Crumetoid form.
(Courtesy of Dr V. Pardo Castella.)

involved. In the red variety the same changes are noted however to a lesser degree. The capillaries in this variety are greatly dilated. No evidence of fungi has been demonstrated. All attempts to inoculate animals have been failures, but accidental and deliberate attempts to inoculate healthy human have been successful.

Symptoms The disease has an insidious onset and is characterized by the appearance of erythematous areas which become achromic. According to Latopi and Blanco, the first manifestation of the disease is an erythemasquamous eruption ("empeines") which is not constantly associated with a positive Wassermann reaction, has been mistaken for an unclassified dermatomycosis, and which disappears under treatment with antimicrobics. In naturally acquired



Fig. 465 Pinta. Late dyschromic stage.
(Courtesy of Dr V. Pardo Castella.)

and its fumes. Intense erythema and desquamation usually precede the desquamation. Many other volatile substances are used in industry which when on a skin subsequently exposed to sunlight result in the production of melanosis at the site of the chemical. Foerster and Schwartz have advised the use of the following for protection against light of those exposed to photosensitizing substances:

Quinine hydroch.	0.2
Stearic acid	29.0
Wool fat	80.0
Ungu. Ipetrolat	50.0
Triethanolamine	3.4
Carbatol	10.0
Terpineol	0.1
Distilled Water	100.0

Pigmentation of Tooth Enamel

Switzer described mottled pigmentation of the enamel of the permanent teeth in a woman twenty-four years of age. This peculiar condition of the enamel was first described in 1910. It involved

the enamel of persons living in areas in which the fluorine content of drinking water was abnormally high.

The fluorine content of water is ordinarily one part per million. It has been shown that deficient diet is not the cause of this condition and that it could be experimentally produced in dogs by the addition of fluorine to their drinking water.

The temporary teeth are rarely affected. The condition appears to occur only in the deciduous molars because enamel of these teeth begins to form after birth.

Only the enamel developing during residence in communities in which the water contains excess fluorine becomes involved. The condition is present on the teeth as they erupt. Some of these communities have changed their water supply and since then their children have had good teeth.

Pink teeth have been observed in porphyria congenita.

PINTA

SYNONYMS: *Mal del pinto*, *carate*, *spotted sickness*.

Pinta is a disease of the skin characterized by pigmentary change. This manifestation occurs without constitutional symptoms.

Varieties. Three stages of pinta are described: the *inflammatory* or *red stage* which is characterized by erythema; the *blue stage* in which the color is produced by enormous numbers of melanin-containing chromatophores; and the *terminal* or *white stage* which is the stage of vitiligo.

Incidence. It is a common disorder of Mexico and Central America but occurs in the whole American hemisphere. It usually affects adolescents of either color. It was apparently a very common disease among the Aztecs.

Etiology. Pinta is a spirochetal infection due to *Treponema herxheimi* (or *carateum*) which produces functional disturbances of the vegetative nervous system. Spirochetes can be demonstrated in scrapings from the lesions, or pustules, as they are called, in a large proportion of cases and patient's serum often gives a positive Wassermann reaction. The *Treponemata* of pinta, yaws, and syphilis are nearly identical and diagnosis commonly depends on the clinical phenomena.

Pathology. In the blue variety of pinta large masses of chromatophores are seen in the papillary and subpapillary layer. Mild inflammatory infiltrations are present, the epidermis remains un-

Pink, minute scars may replace lesions which have completely involuted. There are no subjective symptoms. In some instances, there are scaly lesions which closely resemble those seen in pityriasis rosea. The disease may partly clear



Fig. 467 Pityriasis Lichenoides et Varioliformis Acuta, Female age thirty-four. Condition had been present six months. The lesions were generalized and involved the trunk and upper extremities.

only to relapse several times when new crops of lesions appear. Satenstein sums up the histologic picture. There is an exudative inflammation with parakeratosis, necrotic vesicles on the epidermis which itself shows an extensive invasion of cells, mostly lymphocytes. The cells of the rete show disintegration.

Diagnosis In *maculopapular syphilis* the lesions are more infiltrated, not so bright red, and there are apt to be lesions on the palms, soles, and face. Laboratory and serologic studies are positive for syphilis. Vesiculohemorrhagic necrotic lesions are absent in syphilis. *Pityriasis rosea varicella drug eruptions* and *psoriasis* must also be differentiated. *Parapsoriasis* which is chronic and persistent, shows but one type of lesion a maculopapule, on which the scales are faint but fairly adherent.

Prognosis The general health is unaffected. The disease is self limited run-



Fig. 468 Pityriasis Lichenoides et Varioliformis Acuta (Hahermans Disease)

ning a course of from one month to one year.

Treatment There is no specific therapy. Generalized ultraviolet radiation and a simple calamine lotion are helpful.

pinta the primary plaque is, in 80 per cent of the cases, located on the lower extremities. Generalization occurs two to six months or longer after that. Seropositivity increases in strength as the disease ages. The sites of predilection are the nose, cheek, lower part of the forehead and neck. The bluish pigmentation may also occur in the mouth and beneath the nails. The scalp hair, eyebrows, eyelashes, beard, pubic and axillary hair are unaffected. It may involve the trunk and extremities. Scaling is present in about

one third of the cases. The eruption is symmetrical in about one third of the cases, and the lesions are psoriasiform (in location and appearance), dermatophytoid, syphilitoid, leproid and polymorphous. These pintaids have no specific histologic characteristics. Subjective symptoms are absent.

Treatment. *Antisyphilitic therapy* is effective in treating the blue and red variety of pinta. There is no treatment that has any effect on the areas of achromia.

PITYRIASIS

Pityriasis Lichenoides et Varioliformis Acuta

SYNONYMS: *Parapsoriasis varioliformis* (Wise) *Mucha-Habermann disease*

This is an acute, subacute or chronic generalized eruption in which the lesions consist of macules, papules or vesicles, which show a tendency to crusting, necrosis, hemorrhage and scars. It is rare and not contagious.

Symptoms. The disease is commonest in children and young adults, and appears sporadically. The eruption is multiform, disseminated and symmetrical and appears on the trunk and extremities; mucous membranes are not involved as a rule. The onset is acute and generalized from the outset, as if the result of a toxic endogenous process, and usually without temperature elevation. Occasionally there is generalized adenopathy. The eruption is polymorphous. The lesions may be (1) pigmented or nonpigmented macules; (2) discrete inflammatory or brownish lichenoid papules, which may be round or oval and either with or without fine scales (elicited by lightly scratching the surface of the lesion); or (3) rare vesicles sur-

mounting some papules. Here and there lesions, varicelliform and others not apparently so, are found that have undergone necrosis, crusting and bleeding.



Fig. 466: Pityriasis Lichenoides et Varioliformis Acuta (Habermann Disease)



Fig. 470 Phryniade Rosen. (Courtesy of Dr. Carroll S. Wright.)

Pityriasis Rosen

SYNONYMS: *Pityriasis maculata et circinata*, *herpes tonsurans maculosus*, *pityriasis circinata et marginata*, *Cilbert's disease*

Pityriasis rosea is an inflammatory disease of the skin of the trunk and extremities, characterized by scaly macules and papules varying in color from yellowish rose to salmon

layer. Edema in the epidermis leads to vesiculation and mononuclear leukocytosis. Parakeratosis and acanthosis are evident.

Symptoms The diagnostic feature in pityriasis rosea is the so-called "herald spot" consisting of a red scaly macule upon the trunk and upper part of the limbs. It varies in size from a dime to a dollar and can be elicited in more than



Fig. 469: Pityriasis Rosea. In a female. Note "herald spot" on breast.

Etiology The disease is believed to be due to some infection, although the cause remains obscure. Direct contact with new clothing and with clothing kept in storage is regarded as an etiological factor in some cases. Pityriasis rosea occurs between the ages of ten and thirty years, with an increased incidence in the spring and fall. It is not contagious, and rarely occurs after the age of forty years.

Pathology The histopathology of pityriasis rosea consists of dilated blood vessels, edema, and round-cell infiltration of the papillae and subpapillary

half the cases. Lesions appear suddenly over the trunk, the groins, and upper part of the limbs within ten days after the "herald spot." The face, scalp, palms, soles, and mucous membranes are rarely involved.

Lesions exhibit red follicular papules in some cases (papular form) and the more characteristic pink, oval macules in most instances. They are oval on the thorax with their long axes parallel to the underlying ribs. Lesions range from $\frac{1}{4}$ to $\frac{1}{2}$ inch in length. Pink macules become buff-colored within a few days, wrinkle faintly in the center and peel from their

centers outward. A ring of scales is thus formed with the free edge toward the center and the attached end toward the periphery. The lesions may be vesicular in rare cases and may be limited to one area (localized or abortive form).

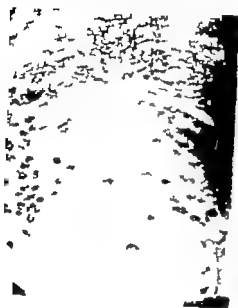


FIG. 471 Pityriasis Rosea. Note the slightly raised oval character of the lesions.

Itching may be severe, slight, or absent. The onset of appearance of lesions is accompanied or preceded, in some patients, by mild intestinal disorder. Adenitis and fever may accompany the appearance of lesions.

Diagnosis. Lesions of pityriasis rosea are confused with psoriasis, macular secondary syphilide, seborrheic dermatitis, tinea circinata, erythema multiforme and copulita rash.

Psoriasis occurs on the distal aspects of the limbs with a history of previous attacks. The scales in psoriasis are silvery and umbilicated. Lesions exhibit peculiar red surface with bleeding point when scales are gently removed.

Macular secondary syphilide is characterized by uniform, round macules without the "herald spot" of pityriasis rosea. Lesions of macular syphilide are less apt to scaling and may appear infiltrated. They appear on the flexor surface of the forearms as well as on the palms and soles. Lesions of the palms and soles rarely appear in pityriasis rosea. Other signs of syphilis, consisting of generalized adenitis, laryngitis, primary sore and a positive Wassermann reaction, are present.

Seborrheic dermatitis occurs over the sternum and between the scapulae. Patches of seborrheic dermatitis are persistent and less regular in shape than those of pityriasis rosea. The face may be and the scalp usually is involved in seborrheic dermatitis and there is evi-



FIG. 472 Pityriasis Rosea. Of primary areas (localized form).

dence in the history of previous attacks. The lesions in pityriasis rosea are symmetrical and the disease is self-limited. **Erythema multiforme** is of more sudden onset and has a wide distribution.

Lesions of erythema multiforme are polymorphous.

The lesions of *tinea circinata* are more inflammatory and have a sharper out-

line than lesions of pityriasis rosea. The center of the lesion in *tinea circinata* is clear and not fawn colored, wrinkled or scaly.

A copaliba rash may resemble early stages of pityriasis rosea. Copaliba rash is of sudden onset, however and more widespread.

The herald spot followed within a week or two by the sudden appearance of lesions is an outstanding diagnostic feature of pityriasis rosea.

Prognosis Lesions clear within three to eight weeks and sometimes even longer. Recurrences (after a year or more) are extremely rare.

Treatment Ultraviolet light given in erythema doses every other day shortens the course of pityriasis rosea. Baths containing barley corn starch, sodium hypochlorite, and potassium permanganate (1:4000) are soothing and of benefit.

Applications of a mild antipruritic lotion are helpful in itching.

Menthol	0.3
Phenol	2.0
Liq. carbonis deterg.	15.0
Icthyol	5.0
Lotion calamine q	150.0

See. Apply with brush four times each day in pruritus.

Sedatives like the bromides and chloral hydrate may be indicated during the onset of pityriasis rosea.

Pityriasis Rubra Pilaris (Deregle)

SYNONYMS *Lichen ruber acuminatus*, *pityriasis rubra follicularis*, *Lichen ruber*

This is a rare progressive, and often persistent cutaneous affection, characterized by the development of tiny acuminate reddish, follicular papules, with or without disseminated, scaly erythematous patches.

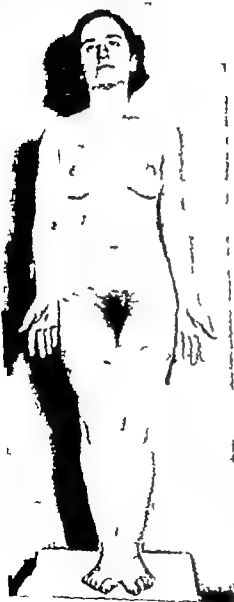


FIG. 475 Pityriasis Rosea. Showing characteristic distribution on face and lower one-half of thighs with the legs spared. (Courtesy of Dr. Jacques P. Geryulere)



Fig. 473: Pityriasis Rosea. Lesion on buccal mucosa (Courtesy of Dr Jacques P Guequierre)



Fig. 474: Pityriasis Rosea. Localized to inner thigh. (Courtesy of Dr Jacques P Guequierre.)

Lesions of erythema multiforme are polymorphous.

The lesions of *trunc circumata* are more inflammatory and have a sharper out-

line than lesions of pityriasis rosea. The center of the lesion in *trunc circumata* is clear and not fawn colored, wrinkled or scaly.

A *copaiba* rash may resemble early stages of pityriasis rosea. *Copaiba* rash is of sudden onset, however and more widespread.

The "herald spot" followed within a week or two by the sudden appearance of lesions is an outstanding diagnostic feature of pityriasis rosea.

Prognosis Lesions clear within three to eight weeks and sometimes even longer. Recurrences (after a year or more) are extremely rare.

Treatment *Ultraviolet light* given in erythema doses every other day shortens the course of pityriasis rosea. Baths containing barley corn starch, sodium hypochlorite, and potassium permanganate (1:4000) are soothing and of benefit.

Applications of a mild antipruritic lotion are helpful in itching.

Menthol	0.3
Phenol	2.0
Liq. carbolic deterg.	12.0
Ichthyol	2.0
Lotion calamine q	150.0
Rec. Apply 4th brush four times each day in periods.	

Sedatives like the bromides and chloral hydrate may be indicated during the onset of pityriasis rosea.

Pityriasis Rubra Pilaris (Devergie)

SYNONYMS *Lichen ruber acuminatus*, *pityriasis rubra follicularis*, *Lichen ruber*

This is a rare, progressive, and often persistent cutaneous affection, characterized by the development of tiny acuminate, reddish, follicular papules, with or without disseminated, scaly erythematous patches.



FIG. 475 Pityriasis Rosea. Showing characteristic distribution on face and lower one-half of thighs. The legs spared. (Courtesy of Dr. Jacques P. Coquerelle.)

Etiology The exact cause is unknown. The disease begins early in life, affects both males and females, and has occasionally shown a familial tendency. The studies of Brunsting suggest a lack

and soles (3) in the form of typical papules on the knees, elbows, forearms, and dorsal surfaces of the phalanges; or (4) in grouped papules upon the body. When the disease develops on the palms, the skin here is red, slightly scaly, dry, and more or less thickened sooner or later, however—in months, sometimes years—typical papules appear



Fig. 476 Pityriasis Rubra Pilaris.
(Courtesy of Dr. Howard Fox)

of utilization of provitamin A or of vitamin A itself as the basic cause.

Aside from the thickening of all epidermic layers, the essential histologic lesion is a hyperkeratosis involving the hair follicle orifices and producing the papule. Secondary inflammatory changes with round-cell infiltration are observed about these follicles.

Symptoms The affection begins in one of several ways: (1) on the scalp in the form of more or less well marked scaly patches; (2) without subjective symptoms and symmetrically as an erythema on the palms or on both palms



Fig. 477 Pityriasis Rubra Pilaris.
(Courtesy of Dr. Howard Fox)

The characteristic papule is discrete, pinhead sized or slightly larger, bright red and firm. It or a group of such papules gives a rough sensation to the pal-

pating finger. The papule is dry and acuminate, and presents at its surface a scale or horny formation, pierced by a hair or showing the end of a broken hair. The papules become more erythematous, and increase in number as surrounding and intervening follicles are involved.

Finally patches usually symmetrical are formed in which the follicular character has faded. These yellowish red, slightly infiltrated patches after a time show only scaly or blackish follicular points, which replace the papules and are covered with fine scales. When such lesions, on the elbows or knees for example are gently curetted, the scales are psoriasisiform. The surface of these patches often presents an exaggeration of the normal fine skin markings. The patches vary in size but are well defined, and at their periphery characteristic papules may be found. Areas of normal skin are constantly found between the erythematous patches. The face, when involved shows a diffuse redness and scaling with ectropion. The nails are yellowish and fragile and often show longitudinal striae.

Diagnosis The typical case, especially in the early stages of the disease, is not difficult to diagnose. However this appears to be one disease in which rigid criteria for diagnosis are best avoided (Wile). *Keratosis pilaris* has different sites of predilection, and the lesions are not acuminate. Acuminate lesions are

seen in *lichen planus* but generally on the body and lower extremities. Typical lichen-planus lesions are, however found elsewhere on the body and in the mouth. Follicular *syrphilides* show a greater tendency to grouping, lack the acuminate horny top, and are apt to be seen on the trunk; there are also other signs and symptoms of syphilis. Abortive cases, in which the disease is limited to the palms or soles, for example or to red scaly patches on the elbows and knees or somewhere on the body are difficult to diagnose and to differentiate from *psoriasis*.

Prognosis The disease is slowly progressive although occasionally it disappears spontaneously. The general health is unaffected.

Treatment A well balanced diet especially rich in carotene is advised. Internally thyroid extract in liberal doses has been occasionally beneficial. Injections of liver extract vitamin B complex intravenously combined with intramuscular injections of vitamin A (100,000 units three times weekly) or vitamin A by mouth (100,000 units daily) over a period of at least three months, should be tried in all cases and especially those showing night-blindness. When vitamin A by mouth fails, a trial of this vitamin is advised. Locally the same therapy used in psoriasis (p. 643) is of value in this disease and especially oil of *codæ chrysarolin* and *pyrogallol*.

POIKILODERMIA

SYNONYMS *Dermatitis atrophicans reticularis, atrophica cutis reticularis cum pigmentatione atrophodermia erythematodes reticularis.*

Poikilodermis Atrophicans

Vasculare

Poikilodermis is an atrophy of the skin characterized by round or irregularly shaped, ill-defined areas of superficial

atrophy of the skin, associated with telangiectases purpuric or pigmented macules.

Varieties The chief varieties of poikilodermis atrophicans vasculare include-

(1) poikiloderma atrophicans vasculare of Jacobi (2) poikiloderma of Civatte (3) poikiloderma congenitale (4) poikilodermatomyxosarcoma and (5) proliferative intertritis

Incidence The disease appears in most instances between the ages of thirty



Fig 178 Poikiloderma Atrophicans Vasculare

and forty five however it has occurred as early as six and as late as sixty years of age. Women and men are affected with equal frequency

Etiology The cause of this disease is unknown. Endocrine pathology may be a causative factor

Pathology The histologic changes depend on the type of lesion examined. (In the early stages the pathological changes are edematous and inflammatory in character and begin around the vessels in the upper portion of the cutis.) The early lesions also show lymphocytic infiltra-

tion around the dilated vessels in the papillary borders and the upper layer of the corium. A definite edema is present; some of the papillae have disappeared, and a rarefaction of the elastic fibers is present, indicating a beginning atrophy. Later the epidermis is thinned with hyperkeratosis and parakeratosis about the follicular orifices. The cells of the basal layer and adjacent rows also show intercellular edema. The individual cells are swollen, the protoplasm stains poorly, and vascular degeneration is present. The nuclei are shrunken and are usually eccentrically located. There is an inversion of the chromatophores. Melanin is also increased in the basal layer. The collagenous tissue is equally affected.

Cana states as follows: "The whole picture gives one the impression of a loose meshed network in which the cellular elements, lymphocytes, connective tissue pigment and mast cells are scattered about."

Symptoms The earliest lesions of poikiloderma vasculare atrophicans are characterized by small round, lichenoid papules, pigmented macules, capillary hemorrhage and atrophy which resembles roentgen ray burns. The skin is dry and pruritus of varying intensity is present. The mucous membranes may be the seat of leukoplakia. Edema and redness of the eyelids have also been observed. The lesions may occur on any part of the skin surface and mucous membranes.

Poikiloderma of Civatte is characterized by reticular pigmentation occurring on the face, neck, the chest and arms. The lesions consist of brown areas intermingled with pigmented superficial and atrophic patches. The eruption is symmetrical. Civatte believed the etiology was due to endocrine disturbance associated with menopause (see p 624).

Poikiloderma congenitale is characterized by telangiectasia, pigmentation, and atrophy, the latter usually following trauma. The condition is present in infancy and a familial history of similar

large atrophic areas; however, myositis and muscular atrophy are associated with the cutaneous symptoms.

Proliferative interstitialitis was described by Barker and Baker. It is an interstitial



FIG. 479. Poikiloderma of Civatte. Eruption consists of reddish-brown patches, intermingled with pigmented, superficial, atrophic, white spots. Telangiectasia is present.

sections is usually obtainable. The mucous membranes are not affected. Thomson reported three cases which showed abnormalities of bone growth, especially of the skull.

Poikilodermatomyositis is quite similar in appearance to poikiloderma vascu-

lar atrophic areas; however, myositis and muscular atrophy are associated with the cutaneous symptoms.

The morphologic picture of this condition has been observed in association with sclerodactylia.

Diagnosis Arcular syphilide, xeroderma pigmentosum, angioma serpiginosum, morphea x-ray or radium dermatitis and lupus erythematosus must be differentiated from poikiloderma vasculare atrophicum. Only *lupus erythematosus* may cause error in diagnosis. In this disease the lymphocytic infiltrate has a definite nodular compact arrangement, while in poikiloderma it has a diffuse network structure. In lupus erythematosus the hyperkeratosis extends over the whole lesion while in poikiloderma only the follicular orifices show a hyperkeratosis with parakeratosis. The cicatricial changes in the epithelium are present in poikiloderma and absent in lupus erythematosus.

Complications The skin lesions of poikiloderma vasculare atrophicum may be followed by ulcerations.

Prognosis Poikiloderma is a progressive disease. It apparently has no effect on the health of the patient, however most of these patients die before reaching old age frequently from leukemia or cancer. Cases have been reported in whom mycosis fungoides has later developed whether coincidentally or not is unknown.

Treatment Endocrine medication might be helpful however up to the present time treatment is of no avail.

Poikiloderma of Civatte

Poikiloderma of Civatte is characterized by reticular pigmentation of the neck (especially lateral surfaces below ears and inframaxillary areas) and face. The chest and arms may occasionally be involved.

Etiology The etiology is not definitely known. It may have an endocrine origin and on the other hand some clinicians believe it to be the result of photosensitization. It occurs especially in females.

Symptoms Lesions are symmetrical and consist of reddish brown patches intermingled with pigmented, atrophic white spots. Telangiectasia is present. In some probably the majority there is only a brownish pigmentation in patches showing a reticulated telangiectasia. Many appear to be of the abortive form with these symmetrical patches in and below the mandibular areas. Subjective symptoms are absent. The condition is more common in women during the menopause.

Diagnosis Poikiloderma of Civatte differs from the *melanosis of Riehl* in that the lesions are limited to the face and neck and occur as a symmetric pigmented and atrophic erythroderma with a retiform arrangement.

POMPHOLYX

SYNONYMS: *Dyshidrosis*, *ketropompholyx*

Pompholyx is an acute cutaneous inflammation limited in distribution to the hands and feet. It is characterized by deeply seated vesicles and bullae.

Incidence Pompholyx occurs frequently between the second and fourth decades of life.

Etiology The etiology of pompholyx remains unknown. Individuals consum-

ing large quantities of tobacco, coffee and tea are more susceptible to attacks. Ringworm is excluded as an etiological factor. Fruit juices, especially orange juice, appear to be provocative in some cases. In a recent study of thirty patients with recurrent vesicular eruptions of the hands, Flood was able to incriminate a food allergen in twenty nine cases. He



Fig. 480 Pompholyx. Note vesicles on fingers.



Fig. 481 Pompholyx.

found the commonest offenders, after extremely strict trial diet, to be tuna fish wheat milk tomato pork pineapple American cheese eggs, lamb chocolate and chicken

Pathology The vesicles are formed in the prickle-cell layer and become filled with serum. Purulence follows pyogenic infection. Inflammatory changes of the derma are slight.

Symptoms Pompholyx is characterized by deeply seated vesicles and bullae accompanied by itching and burning. The vesicles lie deeply in the rete. Vesicles erupt in crops, coalesce and end in exfoliation of the overlying corneum. Secondary pyogenic infection may occur. An attack may last from four to six weeks. Recurrences are common. Lesions have been noted to occur on the conjunctiva along with palmar lesions.

Diagnosis Diagnosis is established by the deeply seated vesicular nature of lesions, their symmetrical distribution and their occurrence in crops. The bilateral distribution of lesions, the brief duration of attacks, and the absence of

fungus differentiates it from *epidermophyton*. Vesiculated dermatoses on the hands, due to chemical contactants, pyrogenic factors and chronic infection with cocci of low grade virulence must also be excluded.

Treatment Vesicles and bullae are opened with a sterile needle. Nonseptic cases respond rapidly to applications of calamine lotion containing 0.5 per cent precipitated sulfur. Septic lesions respond to wet dressings of 1:3000 solution of bichloride of mercury or 0.25 per cent aqueous solution of silver nitrate. Applications of 5 per cent crude coal tar ointment at bedtime are efficacious if lesions have not been irritated. If the lesions have been irritated, 3 per cent *Ichthol* in zinc oxide ointment is indicated. Roentgenotherapy is beneficial in early stages. Arsenic in the form of arsenous acid, $\frac{1}{30}$ to $\frac{1}{4}$ grain administered by mouth three times each day is most efficacious. Alkaline diuretics are also helpful. Possible food allergens should be excluded by a strict dietary control and elimination.

POROKERATOSIS (MIBELLI)

SYNONYMS: *Hyperkeratosis eccentrica* (Reisigl) *keratoderma eccentrica*.

Porokeratosis is a rare chronic affection characterized by slightly atrophic areas limited by a border that is irregularly circular or serpiginous, brownish elevated and hyperkeratotic.

Etiology The condition is seen especially in the male and at any age. It is often familial and hereditary. Bloom and Abramowitz consider it a hereditary keratodyskeratosis. Histologically according to Miescher it is a centrifugally spreading disease of the epidermis leading to porokeratosis with acanthosis and hyperkeratosis, and is not special to the pores which are involved through con-

tinuity of the eccentrically extending disease process.

Symptoms The lesions are chronic painless, and often multiple. They are commonly located on the extremities, especially the hands and fingers, but occasionally on the face, legs, genitals, and buccal mucosa. The elementary lesion is a small elevated horny papule at the pilosebaceous and sweat pores. From the center of the papule projects a keratotic cone sunk into a depression on the papule. As the papules slowly extend centrifugally central atrophy occurs. Finally an annular or serpiginous



Fig. 482 Parakeratosis. Of index finger showing leaved ridges and atrophic centers.



Fig. 483 Parakeratosis (Mibelli) (Courtesy of Dr. David Bloom.)

plaque is produced with a central, smooth hairless area that is defined peripherally by a well marked slightly elevated grayish brown horny linear ridge. The central area is normal skin or has an atrophic appearance or is covered with fine scales. Round oval or irregular gyrate plaques are the usual clinical forms observed. Sometimes the lesions consist of but tiny keratotic wartlike papules or coalescence may form linear lesions resembling a linear nevus.

Diagnosis This is not difficult in well marked cases. When the elevated typical border is absent however diagnosis is difficult and the lesion may then resemble annular lichen planus.

Prognosis This affection is progressive although occasionally it disappears spontaneously leaving a slightly depressed atrophic area.

Treatment Treatment is apt to be unsatisfactory. Excision may be necessary. Electrofulguration of the lesions is beneficial.

PRURIGO NODULARIS

SYNONYMS: *Lichen obtusus cornuus tuberculi cuti pruriginosa, u lichenia perstans verrucosa.*

Prurigo nodularis is characterized by single or multiple itchy nodules on the extremities.

Incidence The disease is met with frequently among adult women.

Etiology The etiology of prurigo nodularis is not known but it is regarded as an atypical nodular lichen simplex chronicus.

Histopathology The histopathology of prurigo nodularis is that of a simple inflammation marked with hypertrophy of the epidermis. The blood vessels in the corium are dilated and infiltrated by lymphocytes and occasional mast cells. The epidermis is characterized by thickening of all layers, particularly of the stratum corneum.

Symptoms The lesions are nodular and verrucous, firm, pea to fingernail in size. They are present in great numbers on the back but are still more numerous over the extremities. The younger lesions have a whitish, pinkish, or brown smooth envelope. As the disease progresses, the lesions become rough and verrucous. Scratching produces fissured and hemorrhagic lesions. The subjective symptom is intense itching. The disease lasts from fifteen to twenty years.

Prognosis The prognosis is unfavorable.

Treatment Treatment is difficult. Lesions may be completely removed by the high frequency current. Some cases respond to roentgenotherapy.

PRURITUS

Pruritus, or itching as an isolated noneruptive cutaneous disturbance is a subjective symptom best described as a sensation in which the urge to scratch or the act of scratching is the essential phenomenon. The only objective evidences of itching are the results of

scratching. Pruritus is a special sense, probably depending on the ordinary sensory nerves for its perception rather than upon special cutaneous sense organs such as are known to exist for touch, pressure, pain, heat and cold. For Torok, itching is a special mode of

activity of the pain apparatus. Itching may be accompanied by sensations of tingling, burning, crawling, smarting, and pricking. It is often intermittent and not infrequently worse at night, to which time it may even be limited. The quality, degree or intensity of the pruritus depends upon the cause but often is largely based on the patient's own interpretation of what he or she feels, and for this the constitutional or nervous makeup of the patient plays a major role. The intensity cannot, therefore, always be judged by the scratching the patient is observed to be doing. In any case however it may actually be so severe as to interfere with the patient's normal conduct and physiological processes, especially sleep.

For practical purposes, the symptom, pruritus, may in general, when discussed as a disease, be considered etiologically (1) as a manifestation of recognized dermatoses; (2) as exogenous and (3) as endogenous, or essential, and this may be either idiopathic or symptomatic.

Pruritus as Manifestation of Dermatoses. Pruritus may precede accompany or follow the appearance of the lesions in such well-known dermatoses as urticaria, eczema, lichen planus, mycosis fungoides, and dermatitis herpetiformis. In those patients in whom the pruritus precedes the characteristic eruption, we are dealing with what has been termed subclinical form of the diseases noted. In the urticarial form, dermatographism may give the clue and the anti-histamines (Benadryl and pyribenzamine) confirm it. The diagnosis in most cases only becomes apparent when the typical eruptive element appears. However, during the noneruptive stage they belong for practical purposes, to the essential forms of pruritus.

Exogenous Pruritus. The exogenous

factors in pruritus are as varied as there are numerous. Pruritus from these agents also varies in intensity and duration with the individual, the nervous irritability and temperament, the presence or absence of systemic disease, phobias, and suggestibility. As Hebra has pointed out, the reaction is a physiological one generally based upon mechanical excitation of the cutaneous nerves or upon some toxic action of the contactant or upon both. Among the exogenous factors are the crawling of insects upon the skin, bites of certain insects, such as fleas, mosquitoes, chiggers, bedbugs, pediculi, and acari; contact with a wide variety of chemicals of animal, mineral or vegetable origin present in or on the clothes or other articles applied cosmetics or cosmesics contacted in one's occupation; uncleanness occasionally but oftener the abuse or excessive use of soap, deodorants, and disinfectants; insecticides, plants and excessively dry skins, both acquired and congenital (xeroderma). In short, all the agents which may produce dermatitis venenata may cause pruritus and pruritus only. In such cases, one might say that the degree of sensitiveness to the contactant is not high, that the dose of the harmful agent in contact with the skin is low or that both factors may be operative. No patient should be classified as presenting essential pruritus until all the exogenous factors have been carefully eliminated. Dermatologists have often observed patients with mild forms of scabies, for example who have been under medical care for many months for nonexistent "acid in the blood." Finally the skin in many of these patients may be normal or merely reddened from rubbing (essentially a traumatic often called "frictional") dermatitis of mechanical origin superimposed upon the pruritus.

Endogenous or Essential, Pruritus
This may be localized or generalized. The localized forms (anus, vulva, scrotum, scalp, nostrils, palms, and soles) will be discussed later. Suffice it to say that, in these, organic disturbances close to the pruritic area are the usual etiologic factors; systemic diseases, however, occasionally require consideration.

In generalized essential pruritus, scratching may leave the skin entirely intact. In others the skin will show linear and follicular erosions, pustules of secondary infection, areas of redness, bleeding points, and small red crusts (dried blood covering an erosion). In others, the skin in localized areas will become thickened to a varying degree (see Lichenification). In some the skin will redden and even thicken under the impetus of the scratching or rubbing, but normal skin rapidly reappears with the cessation of mechanical trauma. It is indeed astonishing how quickly a red, denuded, acutely thickened and spottily eroded skin will become practically normal when scratching ceases as a result of four to five days of complete barbiturate narcosis, occasionally helpful in these cases, or as a result of no scratching or rubbing exhibited by those with sufficient will power and self-control. This is also seen for short periods in psychogenic types, as patients change from doctor to doctor. In some, the scratching is automatic, and the patient is not aware of it or does it while incompletely asleep or dozing.

In a sense the pruritus develops spontaneously and manifests itself by sensations of variable intensity and nature. It may be present in one or several areas and then become generalized or be generalized from the onset. As a rule, more or less frequent exacerbations occur. These may be due to dietary indiscre-

tions, contact with clothing or bed clothes, temperature changes, heat of the bed, violent movements, mental conflicts, and emotional upsets.

In severe cases the need for scratching becomes so urgent that if patients are restrained or cannot satisfy it, they are in agony. Allowed to scratch, they use their nails (which often become extremely shiny and their ends worn down almost to the quick), rough cloths, brushes, combs, metal instruments, anything convenient. In many cases, the itching often stops after the skin has been excoriated—the pain of the excoriation replacing the itching. The patient then often develops a sense of nervous rest and well being.

In mild cases, there may be but one crisis toward evening; in severe cases, a half dozen or more during the night. In all such instances, nocturnal urticaria must be excluded. As a rule when attacks occur during the day and night, it is usually the evening attack which is longest and severest. The duration of essential pruritus varies from several months to one or more years. As a rule, the crises finally decrease in number and severity and eventually disappear.

Essential pruritus may have its inception as a seasonal pruritus during the cold season.

However, it soon loses its characteristics of a winter pruritus, and continues with exacerbations and remissions during the hot season. Occasionally the urine of such patients will show unusual amounts of urates and calcium oxalate crystals, suggesting a gouty diathesis or some protein metabolic disturbance. In most idiopathic types, however, blood and urinary abnormalities are absent.

With essential pruritus are grouped those forms developing under emotional disturbances, which may or may not be

severe but occur in those whose nervous equilibrium is questionable.

Idiopathic Essential Pruritus By far the largest group of sufferers from essential pruritus show no discoverable toxic condition or causative organic lesion. These are presumably persons whose functional disturbance, the pruritus, is the result of repeated insults to the nervous system. This doubtless develops as a result of the hurry and bustle of city life, mental stress and overwork, violent emotion, sorrow, business and financial worries, and abuse of coffee, tea, and alcohol. As a result, a hereditary

calculus in the autonomic nervous system will lead more easily to nervous disequilibrium. It is also believed that the entire vicious circle enhances the chances of disturbances in absorption and elimination. (See also Cutaneous Neuroses, p. 248.)

Senile Pruritus. This form of essential pruritus generally develops after the age of sixty. It is a chronic, incessant—sometimes remittent—torment to the victim. It usually involves the trunk. The remarkable thing about this pruritus is that despite the most intensive scratching there are no or few traumatic cutaneous changes but in most the nails are hung and their ends worn down. The diagnosis, never certain, is made by exclusion. It is believed that senile pruritus is due to peripheral arteriosclerosis or to atrophic and degenerative cutaneous changes incident to age. A diagnosis of senile pruritus is not permissible until the etiological factors discussed under Symptomatic Essential Pruritus (see p. 633) have been excluded.

Senile pruritus generally cedes to no medication. However, generalized ultraviolet radiation and the x rays are occasionally of help. Injections of testosterone propionate (25 mg. twice weekly)

have been advised. Intravenous injections of strontium bromide or calcium chloride (1 gm. three times weekly) may be beneficial. Occasionally the skin is found to be too dry. In these cases, bathing should be infrequent and in tepid water. Soap should be avoided except for intertriginous areas. Superfatted soap like Basis or Shulton's lanolin soap may be used. A soap substitute such as Dermolate may be advantageous. Cotton or silk undergarments are preferable. A cod-liver oil ointment rubbed into the skin twice daily is often beneficial.

Cod-liver oil	40
Petrolatum	80
Lanolin	10
Oleoresin oil, q. s.	

Puncta Prurientia (Itchy Points)

Toomey has described a form of essential pruritus characterized by intense itching at pinhead-sized points of the skin. The itching occurs at irregular intervals during the day and night.

Psychic Communicating Pruritus (Delusion of Itch Influence or Reference) This condition is rare. It is a form of cutaneous neurosis (see p. 248). The chief complaint is not the itching, but its apparent transference to others with whom the patient comes in contact. So vivid is the delusion at times that the slightest move on the part of the observer to scratch while conversing with the patient is proof that even the doctor has become a victim of the patient's pruritus merely by being close to him. In one case, the patient didn't dare sit on her doorstep evenings because she felt she was giving her pruritus to her friends who stopped to chat. In another the patient, when riding in the street car was forced to read a newspaper or keep his eyes fixed upon the floor because he felt the minute he looked at other passengers he caused them to itch.

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With essential pruritus are grouped those forms developing under emotional disturbances, which may or may not be

additional dermatitis from contact with substances to which their skins are intolerant. This situation, when it occurs, complicates the diagnosis because immediately there is an exaggeration of the patient's already existing itch mindedness, and the two situations blend into one another even though the contactant has been eliminated, the other persists to the point where the observer comes to believe a contactant did not add insult to original injury. Certain other factors influencing scratch habit are heredity, mental and physical overactivity or overfatigue, intercurrent ailments or such physiologic states as pregnancy or menstruation, as well as weather changes and heat. The pruritic threshold may be lowered by a variety of emotional factors, such as feelings of inadequacy, anxiety, anger and depression. In some, the relation to sexuality appears certain—the itching and scratching serve to produce sensations akin to sexual gratification. The state of the skin is often given as an excuse to avoid social situations and limit the type of occupation. Practically all of these patients possess a high intelligence quotient, but their lack of an equilibrated nervous mechanism or of a stable involuntary nervous system is suggested by such symptoms as sweating and flushing of the skin on the slightest provocation, frequent subjective sensations of heat and of chilliness, quivering in the abdomen, attacks of diarrhea and sometimes in the normal or red dened skin of small elevations of urticarial aspect, each ranging in size from a pinhead to a split pea.

If memory itch is accepted as a clinical-functional disturbance then the theoretical explanation given by Alvarez on

the mechanism by which nervousness causes discomfort seems applicable to these patients.

The term "homeostasis" has been used by Cannon to indicate the way in which the functioning of the several autonomic organs of the body and the composition of the various body fluids are kept within narrow limits. Cannon has shown that most of this really essential work is done by the sympathetic nervous system which in turn is controlled by a center situated in the hypothalamus. This is further controlled by the cerebral cortex. Cannon studies indicate that when the higher centers are depressed through mental or physical strain there is loss of control of the hypothalamic centers. The suggestion has been made that loss through fatigue or disease the control which the cerebral cortex normally exerts over the hypothalamus is removed then homeostasis becomes so inefficient that the individual becomes painfully conscious of the workings of formerly quiet organs.

Thus a physiologic pruritus becomes exaggerated. The primitive brain of the newborn is but slowly taken control of by the higher centers of the brain. Because of the prolonged nervous excitement—the result of the insomnia and the itching—accompanying the extensive infantile eczema, it is possible that such control was never fully completed in these patients. To state it otherwise, stability of the autonomic, or involuntary part of the nervous system has never been attained. Infantile eczemas carefully treated will prevent and certainly tend to decrease the tendency to the pruritic habit.

SYMPTOMATIC ESSENTIAL PRURITUS.

Physical Allergy WINTER PRURITUS (PRURITUS HEMALIS FROST OR WINTER ITCH). The onset is in the fall of the year. The lower extremities and forearms are the special sites for winter pruritus. The itching generally develops at bedtime and especially on disrobing. Dry skin, excessive bathing, and woolen underwear may precipitate or aggravate the

Habitus Pruriticus (Memory Itch Scratch Habit) This term is used to designate a type of chronic pruritus observed in those who give a history of infantile eczema (p. 329). Close study will show the eczema to have been of the atopic or allergic variety. These patients never have been completely cured of the pruritus which developed with and because of the eczema but which persisted despite the disappearance of the original cause and the eczema itself. It is a well known fact that most if not all infants with atopic eczema acquire at the end of the second or third year a definite tolerance to the food or foods to which they had previously been sensitive or that in any case the eczema has disappeared. This is certainly so for those in whom the manifestation of the hypersensitive state is eczema.

The cutaneous expression of memory itch is usually most marked in some of the areas—especially neck, cubital and popliteal—originally the site of the allergic reaction. The skin is pigmented somewhat thickened reddened at times, and shows linear and follicular erosions and dried blood crusts. The lesions obviously are entirely the result of rubbing or scratching and occasionally of secondary infection—a frictional dermatitis.

Scratching in these patients, one might say is automatic or since the itch sensitivity is marked may follow the slightest somatic psychic or emotional provocation such as exercise or a school examination. These patients scratch at times while conversing with the observer and when told say they didn't realize they were doing it. The lesions under an occlusive dressing or prolonged narcosis disappear but subjective cure does not occur and objective cure is only tempo-

rary. They often go to doctor after doctor improving temporarily with each one. Many patients admit great pleasure from a scratching bout, following which there may be a distinct sense of well-being. Subsequent pain and remorse do not deter repetition of the traumatic act. Herein lies the difficulty of cure: subconsciously many do not apparently desire to be cured.

Practically all of these patients give histories of having been skin tested with all the food allergens—sometimes by several different observers—and although restricted dietetically in accordance with the "positive" skin reactions, have failed to develop the expected clinical improvement or cure. It is a common observation furthermore that the addition of the suspected foods to the diet fails to make the condition worse. Not only are the positive scratch and intradermal tests misleading but so also are the usually positive passive transfer tests. The skin in these patients appears particularly susceptible to trauma a few minutes of rubbing or scratching will damage the skin to a far greater degree than one hour of like trauma applied to a normal skin. Although commonly but erroneously called atopic eczema, the lesions are the result of trauma on a pruritic basis and not those characteristic of eczema. It is a special type of neurodermatitis.

Scratch habit is continuous or irregular but in most patients the tendency to the habit diminishes with time and often disappears entirely generally before the eighteenth year. By this time also they have come to realize that foods bear no relationship to the condition and are well adjusted to a previously held phobia that this or that food is harmful. Occasionally such patients develop an

additional dermatitis from contact with substances to which their skins are intolerant. This situation when it occurs complicates the diagnosis because immediately there is an exaggeration of the patient's already existing itch-mindedness, and the two situations blend into one another even though the contactant has been eliminated, the other persists to the point where the observer comes to believe a contactant did not add insult to original injury. Certain other factors influencing scratch habit are heredity, mental and physical overactivity or overfatigue, intercurrent ailments or such physiologic states as pregnancy or menstruation, as well as weather changes and heat. The pruritic threshold may be lowered by a variety of emotional factors, such as feelings of inadequacy, anxiety, anger, and depression. In some the relation to sexuality appears certain—the itching and scratching serve to produce sensations akin to sexual gratification. The state of the skin is often given as an excuse to avoid social situations and limit the type of occupation. Practically all of these patients possess a high intelligence quotient, but their lack of an equilibrated nervous mechanism or of a stable involuntary nervous system is suggested by such symptoms as sweating and flushing of the skin on the slightest provocation, frequent subjective sensations of heat and of chilliness, quivering in the abdomen, attacks of diarrhoea, and sometimes in the normal or red-dried skin of small elevations of urticarial aspect, each ranging in size from a pinhead to a split pea.

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Thus a physiologic pruritus becomes exaggerated. The primitive brain of the newborn is but slowly taken control of by the higher centers of the brain. Because of the prolonged nervous excitement—the result of the insomnia and the itching—accompanying the extensive infantile eczema, it is possible that such control was never fully completed in these patients. To state it otherwise, stability of the autonomic or involuntary part of the nervous system has never been attained. Infantile eczemas carefully treated will prevent and certainly tend to decrease the tendency to the pruritic habit.

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itching. If the patient remains in a cold climate the pruritus tends to persist the entire winter. Adults, otherwise apparently in good general health, appear to be the chief sufferers.

Treatment consists, when possible, in removal to a warm climate. If this is unpractical then cotton or silk underwear should be worn and an oily preparation such as the following rubbed into the skin morning and evening:

Menthol	0.4
Campbor	1.0
Bitter almond oil	4.0
Sweet almond oil	16.0
Alcohol	16.0
Glycerin q.s. ad	210.0

Daily exposures to ultraviolet and infrared radiation may help. Occasionally the skin is found to be too dry. In these bathing should be infrequent and only in tepid water. Lanolin soap or soap substitutes should be used.

SUNBURN PRURITUS (PRURITUS AESTIVALE) In some people, exposure of any part of the body to sunlight or artificial ultraviolet rays is followed within a short period by itching, sometimes slight erythema of the skin. The symptoms, of this form of physical allergy, persist for from several minutes to an hour unless exposure is prolonged, in which case actual urticarial and eczematous lesions develop or the lesions of *hydra vaccini* form (see p. 411). The relationship between these real cutaneous photophobic states and the various degrees of actinic dermatitis is not clear.

BATH RYTHM In this not uncommon condition the pruritus develops immediately after the person steps out of the tub or shower bath and persists while the skin is wet, while it is being dried, and at times for a variable period after it is dried. In some, it occurs only after the use of hot water; in others, after cold

water and in others the temperature of the water makes little difference. The intensity of the itching varies but may be severe. The lower extremities commonly bear the brunt of the attack. It is generally seen in young adults. Its cause is poorly understood. The condition usually develops suddenly and may persist for months but tends to disappear eventually.

Relief from the itching is frequently obtained by rapidly drying the skin and exposing it to the infrared radiation from an ordinary small electric heater.

Other Causative Factors in Symptomatic Pruritus These are

NERVOUS DISEASES Especially *tuberculosis dorsalis*, *parosis*, and *postencephalitic parkinsonism*.

TOXIC AGENTS Such as (1) drugs, especially opium and its derivatives, belladonna, caffeine, arsenic, quinine, cocaine, strychnine, aconite; (2) foods, especially sea food, condiments, pork and pork products, fermented cheeses, certain raw vegetables (tomatoes, lettuce), alcohol, coffee, tea, tobacco, excess protein intake.

AUTOTOXIC AGENTS Such as those developed as a result of constipation, disturbances of the gastrointestinal tract, diabetes, gout, pregnancy, hepatitis with or without cholemia, clinical and subclinical icterus, mild and severe uremia, chronic nephritis, and anemia.

In many cases of *icteric pruritus* the condition is worse as the jaundice decreases, in which case a disappearing or previously unrecognized jaundice may be detectable only by blood chemical study. In *diabetic* instances, there may be only hyperglycemia. In these the pruritus may persist long after the diabetes has been controlled. In those who have *tuberculosis* the serologic studies of both the blood and the spinal fluid may be

entirely negative and only pupillary and other reflex changes present. In renal disease there may be only an increase in blood-urea nitrogen. In some cases, the treatment of an existing uric-acidemia and the so-called gouty diathesis will clear up the pruritus. It has likewise been noted that generalized pruritus may be one of the earliest symptoms of internal cancer.

FOCAL INFECTIONS. Usually noted are focal infective areas in the sinuses, teeth, tonsils, gallbladder appendix, fallopian tubes, renal pelvis, and prostate. Patients are occasionally cured by the removal of such infected foci. Various authors have also noted syphilis and tuberculosis as factors in the causation of pruritus.

BLOOD DYSCRASIAS. Especially leukemia, pseudoleukemia, lymphosarcoma, and here also may be included the pruritic preeruptive stage of mycosis fungoides.

That certain types of pruritus bear a definite relation to blood dyscrasias, notably the leukemias has been known for a long time. In every persistent case of pruritus, the cause of which has not been determined, a white-cell count and a lymph-gland study including a skin-graphic study of the mediastinal lymph nodes, are indicated. Our knowledge of the relation of essential pruritus to anaphylaxis is too vague for anyone to hazard a definite opinion. Certain it is that generalized pruritus may be the only sign of serum sickness, untoward effects of toxin-antitoxin injections, or ingestion of poorly tolerated drugs or foods (shell fish, cheese, pork and pork products, strawberries, etc.).

OXIDIZ PRURITUS. This is probably of idiosyncratic nature. It is not common and follows the eating of onions, especially raw ones, and their relatives, in-

cluding alcohol. It is often localized to the scalp. It develops shortly after the substance is ingested and may persist for half an hour or longer. Not most applications give relief.

ENDOCRINOPATHY. Endocrine causes have been cited by various authors, especially thyroid and ovarian dysfunction. Itching, prickling, and tingling of the skin have been observed during both the male and female climacteric, and in impotence in the middle-aged male (pychogenic?). In these, testosterone propionate (25 mg., three times weekly for a month) or an estrogen may be valuable.

UNKNOWN EXCITANTS. Among the exciting causes which have been observed as capable of causing a pruritic crisis are many whose interpretation has been very difficult and frequently impossible. This is so for the evening paroxysm, which most pruritics develop at bedtime. In fact, many normal people scratch just as they remove their underclothes, and especially is this true of women. In these cases it is impossible for anyone to say it is cold, contact of air or the removal of pressure which is the excitant. Some of these, however show the clothes-pressure wheals of autographism. One also finds it difficult to explain, except reflexly through the sympathetic nervous system, those cases of generalized pruritus—the indirect result of a localized pruritus—which clear up when the localized pruritus is cured.

Finally it may be said that while pruritus is dignified by a name and usually described as a disease, *per se* it is essentially a symptom of some local or general disturbance of the nervous system, and this disturbance is furthermore secondary to organic and functional disease and even to increased or decreased physiological activities (pregnancy, menopause, age).

Treatment This consists in the determination of the etiological factors and their removal whenever possible. This is generally not difficult for the exogenous forms. For symptomatic and certainly for idiopathic types the problem is not so easy. Symptomatic types may require special medication for the underlying conditions—diabetes, nephritis, pyelitis—together with the local medication indicated in the following. In general, however, measures to include mental rest and dietary restriction are often necessary.

Mental rest means free discussion of the patient's emotional conflicts or change in the patient's environment either by hospitalization or by removal elsewhere. In some extremely prolonged idiopathic types, a permanent change of residence to a warm (Florida) or dry (Arizona) climate has been followed by cure. In any case, a persistent generalized pruritus usually requires considerable patience on the part of both patient and physician. Psychotherapy and reassurance in particular are prime requisites in the treatment of nearly all of these cases. It may be poor consolation to the patient to have explained that itching is physiological and that scratching although the natural response only damages the skin and prolongs the condition nevertheless, there are some among them with sufficient willpower to avoid scratching. Other patients learn that by keeping themselves occupied mentally and physically the severity of the pruritus is partly ameliorated and sometimes to the point where they are not even aware of it until bedtime. Unfortunately, such tactics are helpful to only a few patients.

In some patients, strict limitation of the food intake for several days to a

week is essential for cure. Limiting the diet to rice only or milk only is an excellent procedure in selected cases reinforced later by an alkaline-salt diet.

On the other hand, Foster in cases of *habitus pruriticus*, has obtained excellent results with an acid-ash diet and restriction of fluid intake to distilled water. This diet is reinforced by the internal administration of dilute hydrochloric acid.

In anemia, liver and iron may be indicated. Opium is never given. Sedatives are usually needed but are best avoided as long as possible since they occasionally make these patients worse. Not only may they cause itching but in some they appear to remove the inhibitions or will power still remaining to the patient with the result that there is complete abandonment to the scratching. The sedatives and analgesics generally used are the calcium compounds, bromides, barbiturates, chloral hydrate, carbonyl acetysalicylic acid and coal-tar products (especially aminopyrine). To induce sleep, a hypnotic may be required. It is best combined with an analgesic. Thus, secobarbital, or sodium amytal combined with a coal tar product, usually acts better than one of them given alone (Fantus). If chloral hydrate is used, it is surprising how large the dose must be to obtain results. However, of all the sedatives, chloral hydrate is the one preferred by experienced dermatologists. In some severe cases of complete necrosis, using large doses of sodium amytal may be indicated.

In some cases, Fantus and Cornbleet advise that vasodilators appear of value. The nitrates in the form of glyceryl trinitrate (0.5 mg.) dissolved upon the tongue three or four times a day, ethyl tetrinitrate three times a day

(.03 gm.) for two to three days, or pilocarpine nitrate (8 mg.) three to four times a day for several days, may be tried. The same vasodilating and sedative effects can be obtained by the use of calcium chloride (1 gm.) or strontium bromide (1 gm.) given slowly intravenously daily for five to seven days.

Ergotamine tartrate (1 mg.) three times a day for a week, has been widely used for pruritus of hepatic and renal origin. I jaundice Landy has obtained relief from *procaine hydrochloride* (1 gm. in 500 cc. saline) given intravenously drop by drop for one to one and a half hours. *Colicine* (0.006 mg.) three times daily and the *salicylates* have been found of value in pruritus of gout and hyperuricemia. In the latter *ascotic acid* (50 mg.) intravenously daily for three to five days, has aided.

Nonspecific Therapy The following nonspecifics have all been beneficial at times. intravenous injections daily of *sodium thiosulfate* (1 gm.) *strophanthine bromide* or *calcium chloride* already noted. *Triple-distilled water* in 3- to 8-cc. doses, is also used in the same way.

AUTOMATIC INJECTIONS From 10 to 20 cc. of the patient's blood are removed from the cephalic basilic or other convenient vein, and injected intramuscularly (gluteal or subscapular region). This is repeated at two- three- or four-day intervals for eight to ten treatments.

LUMBAR PUNCTURE (THESMOG) AND OTHER PROCEDURES From 15 to 20 cc. of spinal fluid is removed. In certain of the pure psychogenic types, this is followed by a rapid and prolonged period of relief. One may undertake daily generalized ultraviolet radiation and finally washing of the blood which consists in the removal of 150 to 250 cc. of blood by venipuncture and then its replace-

ment by intravenous injection with 200 to 300 cc. of isotonic salt solution (Fantus and Cornbleet).

Local Therapy Local measures are largely palliative but often give relief for several hours. Protection from all sources of irritation is indicated (wearing of light clothes, obtaining of even temperature, avoidance of hot baths). The chief *antipruritics* are phenol, menthol, and camphor in various percentages in lotions or ointments. Daily bathing and especially the frequent use of soap may be harmful. Even emollient baths (starch, bran, and oatmeal) with or without several ounces of an oil emulsion, are frequently not beneficial but should be tried. Superfatted soaps (Aftershave, Baby, lanolin, Hazeline soap) and soap substitutes (such as acidolate) should be used but infrequently. The following may be of value: *hot applications* applied every two to three hours (plain water with hazel water boric acid water) or *antipruritic lotions* (containing vinegar or camphor [2 per cent] menthol [0.25 to 0.5 per cent], phenol [1 to 5 per cent], lysol [5 per cent], ichthyol [1 to 10 per cent], liquor carbonis detergens [10 to 20 per cent]). In some instances, allowing *hot running water* to flow over certain affected parts, such as the hands for example, may give relief for hours. Sometimes *ointments* are better such as cold cream, petrolatum lanolin cream (water 5 lanolin 5 cholesterolized petrolatum, 10) or cholesterolized petrolatum with 5 to 10 per cent water-soluble Lassar's paste alone or containing 5 per cent ethyl aminobenzoate, 1 per cent camphor menthol, 1 to 3 per cent phenol, and 3 per cent salicylic acid and tartaric acid combined. *Radiation therapy* with x-rays is decidedly helpful in the local forms of

pruritus, with or without secondary changes.

Pruritus Ani, Vulvae, and Scroti

Pruritus ani and pruritus of the genitals is a functional affection of the cutaneous envelopes of these parts and is characterized by itching burning or pricking sensations.

Etiology The etiological factors of pruritus may be any one or more of the following atopic dermatitis, dermatitis seborrhoeicum dermatitis medicamentosa trichophyton or monilial infections, intestinal parasites, food allergy and contact allergic hypersensitivity (toilet paper woolen underwear tight or starched clothing) In some there appears to be a direct relation between the pruritus and seepage from relaxed anal sphincters (Rubinsohn) Pruritus may also result from infection with the colon bacillus, streptococcus fecalis, the staphylococci trichomonas vaginalis toxins produced by bacteria and by skatol (one of the products of intestinal bacteria) Pruritus ani et vulvae may be symptomatic of carcinoma genitourinary diseases, hepatic disease diabetes, pregnancy the menopause and anemia Not infrequently they are psychoneurogenic. A familial history of pruritus ani or allergic disease is often obtainable. Castellani however attributes the lead ing etiological role to fungi

Pruritus ani occurs more often among males (70 per cent as compared to 30 per cent among females) Ninety six per cent occur in those over thirty years of age. It is very rare in pure Negroes.

Pathology The pathological picture depends on the duration and severity of the process. In the beginning there is intense itching with practically no visible lichenification although it is de-

monstrable histologically Later lichenification is present clinically as well as histologically Ulceration may result from excoriations and treatment. The pathology is similar to neurodermatitis.

Symptoms In the early stages, the symptoms are only subjective; that is, itching or burning sensations. Later the skin becomes thickened and lichenified. The skin about the anus presents a grayish or whitish sodden appearance and a foul smelling secretion may exude from the folds. The borders of the area are usually hyperpigmented, while the center of the involved area is depigmented Excoriations and fissures are often present and are the result of scratching Leukoplakia on the mucous surface is a sequela Itching is often sufficiently violent as to produce loss of sleep and as a consequence, nervous exhaustion supervenes

Treatment Roentgen ray therapy probably affords more rapid and more certain relief than any other single mode of treatment. George Andrews recommends that roentgen therapy be started with 200 r (14 E.D.) followed five days later by a dose of $\frac{1}{2}$ E.D. and then after an interval of seven days by a dose of 100 r ($\frac{1}{2}$ E.D.) Following a rest of two weeks, the radiation is continued with 100 r weekly Frequently however x rays are only palliative and since the condition is subject to recurrence, the utmost care is necessary to avoid excessive dosage. While x-ray therapy is being given, every effort must be made to discover and remove the causal factors and to find and adjust topical and other forms of treatment.

Ultraviolet irradiation in suberythema doses every two to four days gives excellent results at times. Before irradiation the skin and anal mucosa

must be cleansed by appropriate measures.

Some patients get most relief from applications of *very hot or very cold water* or in alternations of hot and cold douches. Sitz baths should form an integral part of treatment in all severe cases. The sitz bath is taken *morning and night* or if necessary *three times daily*. Useful solutions are camomile tea, liquor carbonis detergens, solutions of potassium permanganate (1:3000 to 1:20,000) or boric acid solution. When camomile is used, two or three handfuls are placed in a piece of cloth and steeped for twenty minutes in several quarts of hot water then poured into the sitz bath of the proper temperature. Sitz baths should be hot, but not hot enough to burn. The parts should remain immersed for at least twenty minutes.

Local cleansing measures are among the most important procedures. Rubbing with toilet paper often produces violent itching which results either from allergy to the paper or from the friction. Absorbent cotton may be used in place of toilet paper and the parts should be cleansed with a hot boric acid solution after defecation. A sitz bath immediately after the bowel movement is often most beneficial. Patients do better when the prescribed ointment is gently massaged in prior to as well as after defecation.

If much frictional dermatitis is present, it is best to avoid soap and water. Merely apply simple boric acid ointment and cover with a hotex pad. This should be continued for a week or ten days.

One pint of tap water as an *enema* immediately after defecation and at bedtime is occasionally beneficial. Where seepage appears to be a factor the perianal skin should be thoroughly cleansed with soaped water, absorbent cotton

and the parts then dried and dusted with talc.

Local Medication. The type and kind of local medication is determined by the local pathology—whether one is dealing with a dry scaly and denuded surface, or whether the area is moist and macerated, or whether lichenification is present. The scaly scratched, denuded epidermis usually responds best to ointments and *partes* more normal appearing anal regions to *shake lotions*. The moist and macerated perianal and intergluteal regions are best treated with deproteinizing agents, such as silver nitrate, 3, 10, or 20 per cent, or aqueous gentian violet, 1 to 2 per cent, applied twice daily and followed by the application of crude coal tar ointment.

A mixture of partially neutralized *isoparaffinic acids* under the name *Isoper* applied with a finger cot twice daily has been found helpful at times. (Its odor detracts from its extensive use.)

Brown suggests equal parts of 93 per cent phenol and gum camphor applied to the thoroughly dried skin every other day by the physician for two to three weeks. After each treatment, apply boric acid ointment to prevent moisture from contacting the medication which reduces the amount of camphor in the mixture and permits the phenol to burn.

In some cases, half strength *Whitfield's ointment* (3 per cent salicylic acid, 6 per cent benzoic acid in aquaphor) may be used, but the patients should be advised that this ointment will cause burning for five to ten minutes. Five per cent ointment of sulfathiazole is also occasionally of value.

Topical remedies form the mainstay of treatment provided there is no allergic hypersensitivity to the usually effective medicaments. It is always prefer

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Ultraviolet irradiation in suberythema doses every two to four days gives excellent results at times. Before irradiation the skin and anal mucosa

be tried. Results with this procedure have been good in many otherwise refractory cases. The technic is that described in the original report by Hollander. The skin is lathered with liment of soft soap and shaved. Tincture of metaphen is painted on the perineum and washed off with alcohol. Anesthesia is obtained by injection of 20 to 30 cc. of 1 per cent solution of procaine hydrochloride. Phenolated petrolatum is then rubbed into the skin and around the anal canal, to insure antisepsis. The eusnabar is mixed with sterile water to form a thick, syrupy suspension. The instrument used is an electric tattoo machine, with speed of about 3000 vibrations per minute, and a needle handle containing six sterile needles which protrude 2 mm from the machine.

Careful, droplet, subcutaneous injections of absolute alcohol have given relief in some cases.

Surgical removal of the affected skin becomes necessary in the more intractable cases.

Pruritus vulvae

The causes and treatment of pruritus vulvae are essentially the same as those for pruritus ani. Persistent or occasional glycosuria must be excluded in all cases. Because the glycosuria in diabetes mellitus may be intermittent, a blood sugar estimation is indicated in all cases of pruritus vulvae. The psychogenic factor undoubtedly plays an important role in many cases. Some cases of pruritus vulvae are related to abnormalities of the female genitalia and to female hormones, female functions, and hygiene.

Conditions of the female genital tract which cause or irritate pruritus include *Trichomonas vaginalis* vaginitis, monilia infection (aginal onchomycosis or moniliasis) profuse leukorrhea, uncleanness

senile vaginitis, leukoplakia of the vulva and kraurosis vulvae (a progressive sclerosing and atrophy of the mucocutaneous teguments of the vulva). In the first stages of pruritus kraurosis especially the vulvae are red, swollen, eroded and painful. As the lips atrophy they may retain their thick, indurated, leathery appearance on the other hand, there are many cases of pruritus due to kraurosis in which only the atrophy and but slight damage due to scratching are present. The skin and demucosal tissues later become smooth, glistening and parchmentlike. Reflex pruritus of the vulva may result from diseases of the ovaries, uterus, or bladder, and every effort must be made to correct any abnormality of the pelvic organs. Of the general disorders, leukemia and Hodgkin's disease, besides diabetes mellitus, have been causative at times.

The patient must be particularly interrogated from the viewpoint of local irritant or allergic origin of the pruritus. The numerous antiseptic preparations used in douching; the chemical contraceptives including the bases used to hold the active ingredients, deodorant powders and solutions; the chemicals with which sanitary napkins are treated or the materials which they contain; perfumes, creams, and body powder lotions, antiperspirants and deodorants; nail polish; hand lotions; condoms, prophylactic antiseptics, and other substances used by the sexual partner—all these should be investigated as possible causes. The mechanical action of the sanitary napkin may irritate, aggravate and prolong the itching. The sanitary napkin should not be used until the patient is cured. Internal packs being used during menstruation (Tampax).

Treatment. Pruritus vulvae occurs most commonly during the premeno-

able however to combine the use of topical remedies with *physical therapeutic measures* (roentgen rays, ultraviolet rays, etc.)

Turell has obtained good results in intractable anal pruritus by *tattooing* the affected area with *mercuric sulphide*. A simple pneumatic reciprocating pistol is used after patch testing to determine the absence of hypersensitiveness to mercury. The procedure is carried out under local anesthesia and the entire pruritic area is tattooed if recurrence is to be avoided.

Sedation is necessary and recourse to the barbiturates, chloral hydrate and the bromides is recommended.

Patients with pruritus ani are often benefited by a brief vacation at the seashore or in the mountains.

A bland diet omitting all spices, alcohol, hot coffee and hot tea is desirable. A vegetable diet with a small helping of meat daily is advised. Constipation particularly the forcing of hard masses of stool through the canal tends to aggravate pruritus. Mineral oil, cascara milk of magnesia or other mild *laxatives* should be prescribed when necessary.

General measures used in pruritus ani include (1) *autohemotherapy*. Twice weekly 12 to 20 cc of blood is withdrawn from the cubital vein and injected into the buttocks. Equal parts of 0.5 per cent aqueous solution of procaine and the patient's blood serum infiltrated in the affected area weekly for six weeks often effects a cure. (2) Intravenous, intramuscular or oral administration of calcium. (3) Intravenous injection of *strontium bromide* 10 per cent, in aqueous solution of 20 per cent solution of glucose as adjuvant. It should be administered daily for at least ten days thereafter twice weekly if necessary.

The following prescriptions are useful in treating pruritus.

I	
Phenol	4.0
Sol. potas. hydroxide (8 per cent)	4.0
Cotton seed oil	30.0
Sig. Apply as a compress to allay itching.	

II	
Silver nitrat	2.4
Menthol	1.0
Alcohol	30.0
Aqua q.s. ad	100.0

Sig. Apply locally for itching when ecodation and fissures are present.

III	
Camphor	1.0
Phenol	1.0
Benzocaine	1.0
Liq. aluminum acetat	30.0
Cera. albae	12.0
Petrolatum	75.0
Sulf. boratus	1.5
Aqua.	30.0

Sig. Employ as a sedative cream.

IV	
Plumbi acetat	0.12
Liquid t	1.0
Cerate	4.0
Adeps. benzoin	80.0

Sig. For relief of itching due to lichenification.

V	
Menthol	0.5
Phenol	1.0
Spts. vin. rect.	3.0
Pul. talc.	100.0

Sig. Antipruritic dusting powder useful on moist surfaces.

VI	
Benzocaine	1.5
Chlorb. tanol	1.5
Greaseless oint. base (Welcome) q.s. ad	30.0

Sig. Apply locally as occasion requires.

VII	
Benzocaine	1.0
Acidi. tannici	1.0
Hydrag. chlor. mitis	1.0
Lanolin	4.0
Greaseless oint. base (Welcome) q.s. ad	30.0

Sig. Apply locally as occasion requires.

Radical Treatment. If the conservative forms of treatment prove ineffective and when the pruritus is sufficiently severe *tattooing with mercury sulfide* may

be tried. Results with this procedure have been good in many otherwise refractory cases. The technique is that described in the original report by Holander. The skin is lathered with liment of soft soap and shaved. Tincture of metaphen is painted on the perineum and washed off with alcohol. Anesthesia is obtained by injection of 20 to 30 cc. of 1 per cent solution of procaine hydrochloride. Phenolated petrolatum is then rubbed into the skin and around the anal canal, to insure antisepsis. The ointment is mixed with sterile water to form a thick, syrupy suspension. The instrument used is an electric tattoo machine, with speed of about 3000 vibrations per minute, and a needle handle containing six sterile needles which protrude 2 mm. from the machine.

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Treatment. Pruritus vulvae occurs most commonly during the premeno-

pausal or the menopausal period. *Sex hormone therapy* of the menopause often improves the associated pruritus. Nevertheless it is advisable to try the effects of *topical remedies*, roentgen rays, sedation, and other conservative measures, after ruling out allergenic substances and before attempting estrogenic hormone therapy. *Benadryl* has been found to be of value in some cases.

Numerous preparations of the *female sex hormones* (estrogens) are available (stilbestrol a synthetic is a recent favorite). Intramuscular injections of 50 000 I U (10 000 rat units) are given every four to five days until relief is obtained. Smaller maintenance doses are given at longer intervals. The estrogens are also used simultaneously in ointments of 2000 I U (400 rat units) per cc or in vaginal suppositories of the same strength. However they may cause uterine bleeding and large doses of estrogens may even be dangerous because of their possible carcinogenic effects in persons with local or general predisposition to cancerous changes. Furthermore any improvement obtained is lost within four to six weeks after cessation of this therapy.

Therefore a number of courses may have to be administered.

In others, *tattooing with mercury sulfide* and *autohemotherapy* should be tried. Subcutaneous drop injections of 95 per cent alcohol are occasionally beneficial. Failure of all these methods may necessitate *resection* of the pudendal nerves or *vulvectomy*. This latter is undoubtedly the treatment of choice in the presence of chronic leukoplakia or kraurosis vulvae. In these more than half the patients eventually develop cancer.

Pruritus Scroti

Pruritus scroti is usually confined to the scrotum. Only occasionally is the shaft of the penis and perineum simultaneously affected. Fungus infections and dermatitis venenata are the usual causes. Starched clothing, soap, match boxes or suspensories may be the allergens causing an allergic dermatitis of the scrotum. Localized excessive perspiration may excite or prolong scrotal itching. The lesions are those seen in acute dermatitis and the treatment is the same as for dermatitis and other local pruritus.

PSEUDOXANTHOMA ELASTICUM

Pseudoxanthoma elasticum is a symmetrical papular eruption involving the axillae, neck, and flexure folds of the skin.

Etiology. The cause is unknown.

Histopathology. The histopathology of pseudoxanthoma elasticum reveals degenerative changes in the elastic fibers which undergo swelling, granular formation and fragmentation with occasional calcium deposition. Weidman states that this condition is histologically almost identical with some senile elastoses.

Symptoms. Pseudoxanthoma elasticum is a rare skin disease characterized

by the appearance of orange-colored, moist, velvetlike eruptions occurring on the sides of the neck, axillae, abdomen and flexor surfaces of the body. Involvement of the face, scalp, palms, and soles has never been reported.

The papules or nodules comprising this eruption vary in size from a pea to a bean. They are usually discrete but may occasionally coalesce to form striae and patches. The patches and striae may be so wide and long as to conform with the normal creases of the skin in the regions of the neck and axillae. The

integument is usually relaxed, and may appear wrinkled, or it may be in folds. Involvement of the mucous membrane occasionally occurs.

Pseudoxanthoma elasticum is often recognized by the ophthalmologist who discovers angioid streaks on the retina. Involvement of the retina is a distinct early clinical entity associated with this disease. These angioid streaks consist of a reddish-brown band from which glistening streaks extend along the course of blood vessels in the retina.

Elastoma Senilis (Wendman) [*Cutis Rhomboidalis* Vuckar of Miyake] [*Colloid Atrophy* of Daneri]. This is a physiologic state seen particularly in elderly people as part of the general atrophic changes of the skin. It occurs as a diffuse

yellowing on the forehead, cheek, and neck of those constantly exposed to inclement weather. The skin is not only yellow but appears thickened. Wrinkles, as well as normal skin markings, appear exaggerated. The elastic tissue histologically = hyperplastic.

Diagnosis. The absence of xanthoma cells distinguishes it from xanthoma. Differentiation from *colloid milium amyloidosis* and *cutis laxa* is readily made from the concomitant symptoms. In *colloid milium*, a gelatinous material is obtained when the skin is incised.

Prognosis. The disease persists throughout life.

Treatment. Large doses (100,000 units) of *vitamin A* daily might be helpful. No specific treatment is known.

PSORIASIS

SYNONYMS: *Lepros, alphas, psora, Schuppenflechte*

Psoriasis is a chronic recurrent, inflammatory disease of the skin characterized by papules and circumscribed plaques covered by silvery white laminated scales.

Varieties. *Psoriasis Punctata.* This form is a variant of psoriasis, occurring in the form of small, scale-covered papules.

Psoriasis Guttata. This term indicates the form of psoriasis where the lesions approximate the size of drops of water.

Psoriasis Nummularis or *Discoides.* These terms indicate that form of the disease where the patches appear as small coins.

Psoriasis Circinata or *Orbicularis.* This form is characterized by psoriatic patches, with central involution and peripheral extension.

Psoriasis Gyri and *Figurata.* These forms are characterized by spreading circinate patches often producing fantastic figures.

Psoriasis Diffusa. This is that form of psoriasis in which the cutaneous surface is affected in large areas.

Psoriasis Follicularis. This form is characterized by involvement of the coil glands and hair follicles.

Psoriasis Invertata. This form is characterized by deeply infiltrated and often fissured areas which are covered with thick scales.

Psoriasis Inversa. This form is characterized by involvement of the flexures rather than the extensor surfaces, especially inframammary, intergluteal, vulval, and perineal area. The lesions are bright red, smooth, or macerated, and lack the scaly character of psoriasis. Clinically it resembles seborrheic dermatitis; painful fissuring is common.

Psoriasis Ostracea or *Rupoid Psoriasis.* This form is characterized by the presence of oyster shell-like crusts occurring in circinate patches.

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Numerous preparations of the *female sex hormones* (estrogens) are available (stilbestrol a synthetic, is a recent favorite). Intramuscular injections of 50 000 I U (10 000 rat units) are given every four to five days until relief is obtained. Smaller maintenance doses are given at longer intervals. The estrogens are also used simultaneously in ointments of 2000 I U (400 rat units) per cc or in vaginal suppositories of the same strength. However they may cause uterine bleeding and large doses of estrogens may even be dangerous because of their possible carcinogenic effects in persons with local or general predisposition to cancerous changes. Furthermore any improvement obtained is lost within four to six weeks after cessation of this therapy.

Therefore a number of courses may have to be administered.

In others *tattooing with mercury sulfide* and *autohemotherapy* should be tried. Subcutaneous drop injections of 95 per cent alcohol are occasionally beneficial. Failure of all these methods may necessitate *resection* of the pudendal nerves or *vulvectomy*. This latter is undoubtedly the treatment of choice in the presence of chronic leukoplakia or kraurosis vulvae. In these, more than half the patients eventually develop cancer.

Pruritus Scroti

Pruritus scroti is usually confined to the scrotum. Only occasionally is the shaft of the penis and perineum simultaneously affected. Fungus infections and dermatitis venenata are the usual causes. Starched clothing, soap, match boxes, or suspensories may be the allergens causing an allergic dermatitis of the scrotum. Localized excessive perspiration may excite or prolong scrotal itching. The lesions are those seen in acute dermatitis and the treatment is the same as for dermatitis and other local pruritus.

PSEUDOXANTHOMA ELASTICUM

Pseudoxanthoma elasticum is a symmetrical papular eruption involving the axillae, neck and flexure folds of the skin.

Etiology The cause is unknown.

Histopathology The histopathology of *pseudoxanthoma elasticum* reveals degenerative changes in the elastic fibers which undergo swelling, granular formation and fragmentation with occasional calcium deposition. Weidman states that this condition is histologically almost identical with some senile elastoses.

Symptoms *Pseudoxanthoma elasticum* is a rare skin disease characterized

by the appearance of orange-colored moist, velvetlike eruptions occurring on the sides of the neck, axillae, abdomen, and flexor surfaces of the body. Involvement of the face, scalp, palms, and soles has never been reported.

The papules or nodules comprising this eruption vary in size from a pea to a bean. They are usually discrete but may occasionally coalesce to form striae and patches. The patches and striae may be so wide and long as to conform with the normal creases of the skin in the regions of the neck and axillae. The

integument is usually relaxed, and may appear wrinkled, or it may lie in fold. Involvement of the mucous membrane occasionally occurs.

Pseudoxanthoma elasticum is often recognized by the ophthalmologist who discovers angoid streaks on the retina. Involvement of the retina is a distinct early clinical entity associated with this disease. These angoid streaks consist of a reddish-brown band from which glistering streaks extend along the course of blood vessels in the retina.

Elastosis Senilis (Wardman) (*Cutis Rhomboidalis Aechae* of Miyake) (*Colloid Atrophy* of Daneri). This is a physiologic state seen particularly in elderly people as part of the general atrophic changes of the skin. It occurs as a diffuse

yellowing on the forehead, neck, and neck of those constantly exposed to an element weather. The skin is not only yellow but appears thickened. Wrinkles, as well as normal skin markings, appear exaggerated. The elastic tissue histologically is hyperplastic.

Diagnosis. The absence of xanthoma cells distinguishes it from xanthoma. Differentiation from colloid milium amyloidosis and cutis laxa is readily made from the concomitant symptoms. In colloid milium, a gelatinous material is obtained when the skin is incised.

Prognosis. The disease persists throughout life.

Treatment. Large doses (100,000 units) of vitamin A daily might be helpful. No specific treatment is known.

PSORIASIS

SYNONYMS *Lepra, alphas, psora, Schuppenflechte*

Psoriasis is a chronic recurrent, inflammatory disease of the skin characterized by papules and circumscribed plaques covered by silver white laminated scales.

Varieties. *Psoriasis Pruriginosa.* This form is a variant of psoriasis, occurring in the form of small, scale-covered papules.

Psoriasis Guttata. This term indicates the form of psoriasis where the lesions approximate the size of drops of water.

Psoriasis Nummularis or *Discoidea.* These terms indicate that form of the disease where the patches appear as small coins.

Psoriasis Circinata or *Orbicularis.* This form is characterized by psoriatic patches, with central involution and peripheral extension.

Psoriasis Gyrate and *Figurat.* These forms are characterized by spreading circinate patches often producing fan-like figures.

Psoriasis Diffuse. This is that form of psoriasis in which the cutaneous surface is affected in large areas.

Psoriasis Follicularis. This form is characterized by involvement of the coil glands and hair follicles.

Psoriasis Inversata. This form is characterized by deeply infiltrated and often fissured areas which are covered with thick scales.

Psoriasis Inversa. This form is characterized by involvement of the flexures rather than the extensor surfaces, especially inframammary, intergluteal, vulval, and perineal areas. The lesions are bright red, smooth, or macerated, and lack the scaly character of psoriasis; clinically it resembles seborrheic dermatitis; painful fissuring is common.

Psoriasis Ostracea or *Rupoid Psoriasis.* This form is characterized by the presence of oyster shell-like crusts occurring in circinate patches.

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interrupted by psoriasis. Baldness never occurs. The palms and soles are rarely involved, but the nails are frequently affected. The diagnosis of psoriasis of the nails is established by the coexistence of psoriatic lesions elsewhere on the body. Involvement of the nails calls

and give a history of long-standing psoriasis.

Palmar and plantar psoriasis may be limited to either palms and soles or it may affect both palms and soles together. It is characterized by sharply defined, localized small papules, or large more or less erythematous plaques. The lesions may be bright red, yellowish brown, or hyperkeratotic with deep fissures. Pustules or small vesicles may be associated.

The glans penis may be the site of psoriasis where it is characterized by



Fig. 485 Psoriasis.

for microscopic examination of the tissue to rule out fungus infection. The characteristic nail changes are stipping, brownish discoloration, lifting of the distal portion of the nail from its bed, cracking of the free edge of the nail and the formation of crusts beneath it.

The mucous membranes are rarely involved.

Schanberg has also divided psoriasis into three stages (1) eruptive (2) quiescent, (3) spontaneous decline and (4) disappearance.

Aberrant Type. Pustular psoriasis is rare and is characterized by the presence of pustules within the psoriatic plaque. The patients are usually febrile



Fig. 486 Psoriasis.

erythematousquamous lesions. In this location it resembles balanitis, lichen planus, or erythroplakia of Queyrat.

Erythroderma psoriaticum is characterized by the presence of a generalized erythema. It arises in connection with

Psoriasis verrucosa This form is characterized by an excessive thickening of the patches to such an extent that they have a warty appearance

Pustular Psoriasis This form is characterized by the presence of pustules they may be an essential part of the disease or they may develop in the course of the disease The palms and soles are the usual sites of this variety

In the *arthropathic form* of psoriasis, arthritis and rheumatoid symptoms are present

Etiology The cause of psoriasis is unknown Some authors have contended it to be an "id" manifestation of a fungus infection or that it is due to an organism on the skin surface It may be the result of faulty metabolism or is perhaps a symptom of neurocirculatory instability Köbner called attention to the occurrence of psoriatic lesions at sites of irritation This is referred as *Köbner's phenomenon*

Most cases begin between the ages of five and fifteen It occurs with the same frequency in both sexes About 30 per cent of patients give a history of psoriasis in antecedents

The nitrogen balance is disturbed in psoriasis (Schamberg et al)

Pathology The pathology of psoriasis varies according to the stage of the disease and location of lesions

A considerable degree of *parakeratosis* is present in early lesions The stratum mucosum is thickened and infiltrated from intercellular and intracellular edema Microabscesses filled with polymorphonuclear leukocytes are often found Processes of the rete malpighi are elongated while the epithelium covering the tips of papillae is thin The papillae are edematous and their vessels are dilated A moderate perivascular round cell infiltration of the papillae

and superficial dermis is present The silver appearance of scales is due to the presence of linear air spaces in the horny layer

Symptoms The primary lesions of psoriasis are erythematous flat papules covered with silvery white scales The



Fig 484: Psoriasis Gyrate et Figurata.

primary papule grows by peripheral extension and coalesces with neighboring papules to form a plaque The plaques are covered by abundant silvery white lamellated scales The psoriatic lesion is never vesicular and is not followed by scar formation Tiny bleeding points (dew drops) appear on forcible removal of scales

The common sites of predilection include the scalp, elbows, knees, and sacral region Circinate gyrate, and serpiginous figures are formed on the trunk as a result of peripheral extension of lesions and the tendency to central healing The scalp may be involved in its entirety or in the form of several circumscribed scaly patches The growth of hair is not

interrupted by psoriasis. Baldness never occurs. The palms and soles are rarely involved but the nails are frequently affected. The diagnosis of psoriasis of the nails is established by the coexistence of psoriatic lesions elsewhere on the body. Involvement of the nails calls

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Erythroderma psoriaticum is characterized by the presence of a generalized erythema. It arises in connection with

overmedication intravenously given at senicals in particular and the intolerance to local therapy

Arthropathic psoriasis is accompanied by polyarthritis. The joints of the hands and feet are more often involved although all joints may be affected. Arthropathic psoriasis is more

common among women. More than 50 per cent of patients with arthropathic psoriasis show involvement of the nails. The arthritis tends to appear with each attack of psoriasis.

In many patients, psoriasis—partially or wholly—disappears in summer only to return in cold weather. In a smaller



Fig. 487: Psoriasis

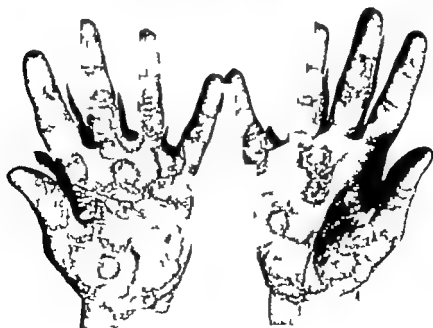


Fig. 488: Palmar Psoriasis. (Courtesy of Dr. Ralph Bernstein.)

number of cases, the reverse is true. Subjective symptoms are usually absent, although in the acute cases burning or smarting of the skin is present.

Diagnosis. Psoriasis is differentiated from seborrheic dermatitis, pityriasis rosea, lichen planus, eczema, syphilis, and lupus erythematosus. In psoriasis, all the lesions, whether small or large, have the same characteristics. In psori-

By methodical grattage, serous ooze (not hemorrhagic puncta) develops.

The eruption in *pityriasis rosea* is limited to the trunk, upper arms, and thighs. Its onset is characterized by the "herald spot, which is an oval, brownish yellow lesion covered with fine scales. This "herald spot" is succeeded within about ten days by numerous smaller lesions. The duration of *pityriasis rosea*



Fig. 489. Palmar Psoriasis.

as also, under very careful, gentle curet- tage a typical psoriatic lesion will first shed the scales, then a smooth, fine pel- licle, and then, as the observer continues to curet, there develop fine punctate hemorrhages as the elongated papillae are traumatized. This last diagnostic sign is best observed if cigarette paper under a glass slide is pressed down on the lesion. The whole procedure is known as methodical grattage of Brocq. Seborrheic dermatitis commences invariably on the scalp. Its scales are oil and few in number. The axillae and flexor surfaces are involved. The histologic picture in seborrheic dermatitis is characterized by mono- nucleous vesiculation, and spongiol-

is about two months. The condition disappears without treatment.

Lichen planus appears chiefly on flexor surfaces of the forearms, wrists, and ankles. The patches are pruritic, often thickened and of violaceous color. They are covered by a horny film rather than by scales. Individual papules comprising a patch are polygonal in outline.

Eczema has a predilection for flexor surfaces. Its lesions are not sharply defined, and are often moist and very pruritic.

Populosquamous syphilis occurs principally on the palms and soles which are rarely involved in psoriasis. The lesions of syphilis are infiltrated dark

overmedication intravenously given arsenicals in particular and the intolerance to local therapy.

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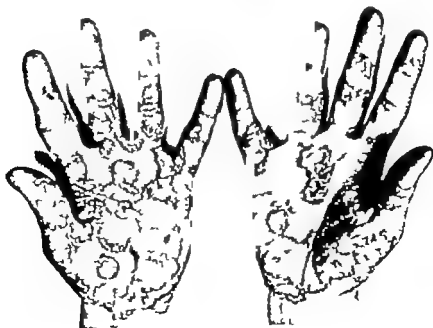


Fig. 488 Palmar Psoriasis. (Courtesy of Dr. Ralph Bernstein.)

ans but of these only a few are of value.

Arsenic is beneficial in suitable cases. It is indicated when the disease has been present for some time, and the spots are pale and have ceased to spread. Arsenic and all stimulating applications are contraindicated when the disease is recent, and the spots are red and spreading. For those, autohemotherapy at five-day intervals is the preferable procedure. Fowler's solution remains the favorite arsenical for oral administration in doses of 5 to 10 drops three times each day after meals. The course of arsenic treatment ranges between two and three months. It is not repeated until several months of rest have elapsed.

Arsenical injections are often preferable to oral administration. Salzberger and Wolf prefer subcutaneous injections of a 2 per cent solution of sodium arsenate containing 1 per cent phenol.

Sodium arsenate	20
Phenol	10
Aq. distillat. q. ad	1000
M Dispense in sterile bottles in rubber stoppers	

The initial dose is 1 mmim. It is increased by 1 mmim each day until 30 mmims are given.

Arsenical treatment is discontinued on the appearance of gastrointestinal cramp, diarrhea, conjunctivitis, edema of the eyelids, and when such dermatoses as erythema, lichen planus, herpes zoster, herpes simplex, purpura, itching, etc. are evident.

Sodium salicylate is of undoubted value in suitable cases. It should be given in solution rather than in tablet form. It is indicated when the disease is acute and characterized by the appearance of red spot and spreading lesions.

Potassium iodide is applicable in all stages of psoriasis. It is administered

in doses of 4 gm (1 dram) three times each day.

Thyroid is probably useful in patients past middle age. Small doses of thyroid when combined with small doses of arsenic produce better results than when either drug is given alone.

Large doses of vitamins A and C have been of value in some cases. Massive doses of vitamin D have advocates. Brunsting prescribed daily doses of 100,000 units vitamin D concentrate. Vitamin B₁ (thiamin hydrochloride, 1000 international units daily) a low fat diet and an exfoliative paste have produced favorable results.

The intravenous injection of autogenous vaccines has been suggested for psoriasis. Sutton employed with satisfaction large doses of autogenous colon bacillus vaccine together with local applications of 20 per cent chrysarobin ointment in chronic cases. Weekly injections of 10 cc of the patient's whole blood into the buttock are helpful in the acute variety of the disease.

The diet should consist essentially of vegetables, lean meats, and fruit. It should not contain more than 20 gm of fat for each day. Patients may eat fat free soups, lean meats, all fruits, fruit juices, starches, and all vegetables and prepared fat. Fat soups, fat meats, sausage, fat poultry, fat fish, cream, cheese, clabber, maize oil, corn oil, gravies, and alcohol must be avoided.

External Treatment. *Chrysarobin* and *anthralin* are the most efficacious remedies, but they are not always well tolerated, and give an irradicable violaceous color to the skin. *Chrysarobin* also stains the clothing and, because of this, is by some preferably applied in colloidal

Chrysarobin
Flexible colloidal

40
300

red or copper-colored and arranged in serpiginous outline. Concomitant signs of syphilis consisting of palpable lymph nodes, mucous patches, and a positive serology are usually present.

The scales of *lupus erythematosus* are gray and adherent. Lesions occur usu-



Fig. 490. Psoriatic Erythroderma. The entire skin is red and thickened. The condition followed an arsphenamine injection for psoriasis. Discrete psoriatic lesions are still present despite the generalized dull redness of the skin which had been present for six years.

ally on the face and the scalp where atrophy and alopecia are present. The follicular openings are patulous due to projecting plugs within follicles.

Complications. Complications usually develop as the result of treatment. Local medication when applied in the acute stage of psoriasis may convert the eruption into exfoliative dermatitis. Dissemination of the eruption, the production of eczema or erythroderma, may result from the use of medication to which the patient is hypersensitive such as the *tars mercurii chrysarolin*, etc.

Prognosis. Psoriasis does not interfere with one's health and permanent relief of a severe case of psoriasis cannot be promised. The prognosis of individual attacks of psoriasis is good but recurrences are the rule. The frequency of recurrence varies with individuals; it may recur repeatedly during the patient's lifetime. Lesions of short duration are more amenable to treatment than those of long standing. It is important to institute prompt treatment. The prognosis is hopeless among alcoholics. The prognosis of psoriasis is more favorable if the patient can be removed to a tropical climate.

Treatment. Therapy should depend upon the stage especially although site, severity and location all influence the therapeutic procedure to be adopted.

In the acute eruptive stage, active medication is apt to be harmful and should be limited to the following: (1) diet of fruit and vegetables; (2) alkalization with liq. potassii 1 to 5 drops, well diluted in water; (3) autohemotherapy every third day for three to four weeks, and (4) vaseline locally.

In the quiescent or chronic stage, the following procedures are indicated.

Systemic Treatment. A great many drugs have been used for treating psor-

advisable to apply a second coating of the ointment on the first one before retiring for the night. An inexpensive cotton suit of underwear or a gauze covering is worn to protect the bedclothing.

The technic of autohemotherapy consists of withdrawal of 10 cc. of blood from the cubital vein, immediately injected into the buttocks, 5 cc. on each side. The procedure is repeated five or more times at two- to three-day intervals.

For hospitalized patients with generalized psoriasis, in which the lesions are chronic, numerous, and thickened, a method of therapy that is often successful consists in a *low-protein diet* (not more than 100 calories daily) together with the Cade bath (Emboutaud) which may also be used for ambulatory non-hospitalized patients.

Oleum cod. (R.B.)	20
Oil Cade	1000
Yolks (2 eggs)	
Flouider Phosphorus q. emulsion	
Acid Chrysocollum	20
Aq. Dest.	1000

Add the 1000 cc. to hot bath in which patient remains one hour. Rub skin and even scalp constantly (but protect eyes) with this medicated water while in the bath, use neither brush nor soap. The skin must be watched for signs of chrysa robin dermatitis. Repeat bath daily for 5 or 6 days and stop the following two days. Good results appear by third week although baths may have to be continued four to 6 weeks; after lesions have disappeared one bath weekly or every other week is sufficient. Hair (in female) should be thoroughly dried and strands

wiped with cotton dipped in compound spirits of ether. This helps remove the tar odor. To reduce cost, the same bath may be used three or four days.

Although the association of arthritis and psoriasis is not substantiated by evidence other than empirical, the arthritic symptoms occasionally accompanying psoriasis have been known to subside when skin lesions disappear. Arthropathy resisting psoriatic treatment may be improved by use of *foreign proteins* *heat* and *massage*.

The treatment as outlined has permanently checked the disease in 15 per cent of 2000 cases; in another 15 per cent, the plaques returned within two to three months. The routine is safe and may be resorted to as often as necessary without fear of complications.

Baths of copper sulfate (0.1 per cent aqueous solution) followed by the application of the following, has been most helpful.

Vioform (Ciba)	1.5
Ung. petrolat.	60.5
Sig. Apply with massage daily	

Up to the present time no drug has been proved to have specific value in psoriasis. Occasionally one or a combination of several of the following drugs has been beneficial. Tara, ammoniated mercury calomel, chrysarobin, anthralin pyrogallol, sulfur acid salicylic, and resorcin.

If treatment is to be successful, alcohol, coffee, tea, and all other stimulants are best avoided. A low fat diet has also been extolled.

Paint each psoriatic lesion allow to dry and then apply another coat of plain flexible collodion as additional protection for the clothing

The following prescription containing chrysarobin is definitely helpful

Chrysarobin	20.0
Oil ricin.	20.0
Acid salicylic	10.0
Petrolatum	50.0

Sto. Apply twice each day for two weeks.

Becker and Obermayer believe that a 5 per cent ointment of crude coal tar is the best treatment for psoriasis. The ointment is vigorously rubbed into lesions morning and night. The skin is then irradiated with ultraviolet light in gradually increasing doses following removal of ointment.

A 50 per cent ointment of calomel has been beneficial.

Psoriasis of the scalp is treated by an ointment of ammoniated mercury with or without salicylic acid supplemented by frequent shampoos of green soap.

Hydrarg. ammoniat.	2.0
Acid salicylic	2.0
Liq. carbonis detergens	3.0
Oil ricin.	6.0
Petrolatum q.s.	60.0

Sto. Apply at bedtime Shampoo scalp very morning with tar

Kilroy advises the frequent application of the following

Hyd. chlor. corr.	0.1
Acid. salicylic	} 5-15% of each
Acid. lactic	
Acid. acetic	
Formalin	
Sp. vini. rect. q.s.	240.0

Röntgen therapy administered in $\frac{1}{4}$ erythema doses once a week for four weeks is helpful in treating small areas of psoriasis. It is especially valuable for psoriasis of the nails, palms, and

soles. Exposure to the sun is definitely beneficial.

Patients are impressed with the importance of continuing scalp treatment long after all signs of the disease have disappeared. These areas are to be watched for signs of new attacks.

Ultraviolet therapy in erythema doses is beneficial in some cases, while in others it is valueless and even precipitates an additional outbreak.

Goeckerman in 1943 introduced an effective treatment which consists of the use of coal tar ointment. The ointment consists of crude coal tar 12 or 24 pulverized zinc oxide, 12, and corn starch 30 in petrolatum 60. Adjuvants are ultraviolet light, oatmeal baths and autohemotherapy. On the first day in the hospital all the patches of psoriasis are covered thickly with the ointment, which is left on overnight. The following morning it is removed with a light weight mineral oil care being taken to leave a thin film of the oil on the skin. The reason for this is not known but it is essential paradoxical as it may seem, in view of the fact that ultraviolet light therapy is given to the skin through the film of oil. At the first treatment the light is applied for one minute at a distance of 30 inches, after dividing the skin surface into six areas. The time of exposure is increased and the distance decreased each day in order to maintain a state of mild erythema.

After the light treatment the patient spends half an hour to two hours in an oatmeal bath or in an ordinary tub of water kept at approximately 95° F. which loosens the scales and allows removal by brisk rubbing of the skin while in the tub. After the bath a thick coating of the ointment is applied again to the skin an endeavor being made to put it mainly on the patches. It is usually

advisable to apply a second coating of the salve over the first one before retiring for the night. An inexpensive cotton suit of underwear or a gauze covering is worn to protect the bedclothing.

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Odorless oil (R.B.)	50
Oil Cade	100 g
Yolk (8 eggs)	
Flouider Protein 9	emulsion
Acid Chrysophanic	50
1/2 Doal.	1000 g

Add the 1000 cc. to hot bath in which patient remains one hour. Rub skin and even scalp constantly (but protect eyes) with this medicated water while in the bath, use neither brush nor soap. The skin must be watched for signs of chrysarobin dermatitis. Repeat bath daily for 5 days and stop the following two days. Good results appear by third week although baths may have to be continued four to five weeks; after lesions have disappeared, one bath weekly or every other week is sufficient. Hair (in female) should be thoroughly dried and strands

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Vioform (Ciba)	15
Ung. petrolat.	50.0
Bat. Apply 1th massage daily	

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If treatment is to be successful, alcohol, coffee, tea, and all other stimulants are best avoided. A low fat diet has also been extolled.

PURPURA

SYNONYMS: *Haemorrhoea petechialis, hemorrhagies cutaneae.*

Purpura is a disease of the blood or capillaries characterized by red and purple cutaneous patches resulting from multiple hemorrhages within the skin and mucous membranes. The condition may be precipitated by drugs or infection (asymptomatic or secondary purpura)

of hemorrhagic macules on the legs. The thighs and forearms are occasionally involved. The macules are definitely circumscribed and are of red or purple color. The disease runs its course in two or three weeks. Recurrences are common and may extend over a period of



FIG 491: Purpura. (Courtesy of Dr. Carroll S. Wright.)

Purpura may also be a primary symptom (idiopathic purpura)

Varieties Several clinical forms of primary purpura are recognized: purpura simplex, purpura rheumatica (pelliosis rheumatica of Schönlein), Henoch purpura, and purpura haemorrhagica (morbus maculosus of Werlhof). Hemorrhagic diseases are also classified pathogenically (platelet deficiency, coagulation disorder, capillary angiosis).

Purpura Simplex

This is a mild form of the disease characterized by the sudden appearance

years. Constitutional symptoms are absent. Associated constitutional symptoms are mild.

Purpura Rheumatica

SYNONYMS: *Pelliosis rheumatica, Schönlein's disease.*

This has the same distribution as purpura simplex but is always associated with arthritis. The condition starts with an angina accompanied by a temperature of 100° to 103° F. Lesions are often limited to the neighborhood of affected joints. The arthritic pains coincide with the development of lesions. The petechiae

may be accompanied by urticarial, erythematous, nodular and bullous eruptions which are not unlike those of erythema multiforme.

Pericarditis, endocarditis, and pleurisy may be associated with purpura rheumatica.



Fig. 492 Purpura Simplex.

Henoeh's Purpura

This occurs in early life and is characterized by recurrent purpuric attacks associated with abdominal symptoms which include colicky pains, vomiting and diarrhea. Vomiting of blood and the passage of bloody stools may occur. Nephritis, hematuria, and arthritis are sequelae of Henoeh's purpura. The cause is ascribed to allergy to foodstuff.

Purpura Haemorrhagica

SYNONYMS *Morbus maculosus Werthoffi*, *thrombopenia essentialis*.

Purpura haemorrhagica is of sudden onset and is characterized by extravasation of blood from mucous membranes in addition to the presence of cutaneous lesions. The nose, the mouth mucosa, including the gums, the intestines, stomach, uterus, and the genitourinary tract are common sites of hemorrhage. Cutaneous lesions consist of small, dark, pinpoint-sized hemorrhages (petechiae) and larger, irregular, purplish-red patches (ecchymoses) which may coalesce.



Fig. 493 Purpura Haemorrhagica Bullosa (Crocker)

PURPURA

SYNONYMS: *Haemorrhagica petechialis, hemorrhagias cutaneas.*

Purpura is a disease of the blood or capillaries characterized by red and purple cutaneous patches resulting from multiple hemorrhages within the skin and mucous membranes. The condition may be precipitated by drugs or infection (symptomatic or secondary purpura)

of hemorrhagic macules on the legs. The thighs and forearms are occasionally involved. The macules are definitely circumscribed and are of red or purple color. The disease runs its course in two or three weeks. Recurrences are common and may extend over a period of



Fig 491: Purpura (Courtesy of Dr. Carroll S. Wright)

Purpura may also be a primary symptom (idiopathic purpura)

Varieties Several clinical forms of primary purpura are recognized: purpura simplex, purpura rheumatica (petiosis rheumatica of Schönlein), Henoch's purpura and purpura haemorrhagica (morbus maculosus of Werlhof). Hemorrhagic diseases are also classified pathogenically (platelet deficiency, coagulation disorder, capillary angrosis).

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other types of purpura. No specific internal remedy is indicated. Diluted aromatic sulfuric acid is given in doses of 1 to 3 cc. Calcium chloride calcium lactate and oil of turpentine have also been advised. Ascorbic acid, injections of thromboplastin and snake venom are of occasional value. Blood transfusions are indicated in severe cases. Splenectomy has been followed by permanent recovery in purpura hemorrhagica.

The following formulas are recommended

I

Solution of epinephrine hydrochloride
 1 cc. 0.5 to 0.6 cc. intramuscularly three times each day (indicated in hemorrhages from the mucous membranes)

II

Sterile solution of calcium chloride-urea
 1 cc. 10 cc. twice every 10 days for six doses

Purpura Annularis Telangiectodes

SYNONYMS *Majocchi's disease*
follicularis annularis, telangiectoma.

Majocchi's disease is a rare type of purpura characterized by telangiectatic, purpuric and atrophic lesions on the lower extremities including occasionally the thighs, the forearms, and trunk.

Incidence The disease occurs more often in young male adults and only occasionally in children.

Etiology The cause is not known, but it is probably a vasomotor phenomenon.

Varieties Ma Kee divides the manifestation into three stages telangiectatic, purpuric and pigmentary and atrophic.

Pathology The characteristic histologic feature consists of an obliterative endarteritis with hyalinization and occlusion of vessels of the deep reticular layer and the subcutaneous tissue and



Fig. 494 Purpura Annularis
 Telangiectodes.



Fig. 495 Purpura Annularis Telangiectodes (Synonym Majocchi's Disease)

neous hemorrhage may be petechial or ecchymotic and may appear spontaneously or follow trauma. The disease may be acute or chronic. It commonly begins during childhood and occurs more often among young girls. The first symptom consists of weakness followed within a few days by rapidly increasing purpuric lesions. The skin presents a characteristic mottled appearance which is followed by hemorrhage from mucous membrane. It lasts from two to eight weeks and may be intermittently present for many years.

The constitutional symptoms consist of fever, arthritis, vomiting, headache and prostration. Anemia of the brain may result. Death usually occurs from anemia from brain hemorrhage or hemorrhage within the suprarenal glands.

The blood picture is characterized by considerable reduction in platelets. The platelets may be reduced to 1000. Bleeding is prolonged. The clot is soft and nonretractile. Clotting time is normal.

Purpura Fulminans. This is a form of purpura haemorrhagica occurring among children following scarlet fever. It is usually fatal.

A variety of idiopathic purpura is often seen among the aged and referred to as "purpura senilis." The lesions of purpura senilis occur on the legs and are dependent on senile vascular changes. An attack of purpura senilis may occasionally extend intermittently over a period of years and is referred to as "chronic" purpura. The skin and mucous membranes are normal during the quiescent period.

Symptomatic Purpura

Purpura may occur in typhus fever, cerebrospinal fever, typhoid fever, scarletina, measles, variola, vaccinia, pyemia, septicemia, bacterial endocarditis, eryth-

ema multiforme, dermatitis medicamentosa, serum sickness, and herpes zoster. It may also occur in Rocky Mountain spotted fever, malaria, anthrax, influenza, general tuberculosis, and syphilis.

Deficiency of vitamin C increases the fragility of the capillaries and accounts for the purpura occurring in connection with scurvy.

Bites, especially the flea bite, may lead to purpura. Cullen's sign is purpura occurring about the umbilicus due to a ruptured ectopic pregnancy.

Toxic Purpura

The administration of iodine and the iodides, snake venom, mercury, antipyrine, chloral hydrate, copaiba, benzol, arsenicals, gold, phosphorus, quinine, ergot, turpentine, belladonna, and the salicylates may produce purpura.

Menstrual purpura has also been reported. The rash in menstrual purpura involves the lower extremities and may be accompanied by febrile reaction.

Nervous purpura may follow severe fright, neuritis, and the painful paroxysms of tabes.

Prognosis. All patients with purpura, except those with the fulminating hemorrhagic, and septicemic forms, recover without difficulty.

Treatment. The treatment of purpura depends upon the underlying cause. Thus, a hemorrhagic tendency may be due to a deficiency in prothrombin, an essential component of the clotting process. This occurs in obstructive jaundice, extensive liver disease, sprue, ulcerative colitis, and hemorrhagic disease of the newborn. Parenteral administration of vitamin K will control the hemorrhagic states due to hypoprothrombinemia, except that associated with hepatic disease.

Simple purpura requires no treatment. Absolute rest in bed is essential in the



Fig. 496 *Purpura*. (Courtesy of Dr. Carroll S. Wright.)

a round-cell infiltration. The arteries and veins are involved in the process.

Symptoms. The early lesions are sharply defined rose-colored telangiectatic then purpuric macules. They enlarge by peripheral expansion until they attain a diameter of about 2 cm. Enlarged patches gradually heal in the center. Coalescence of expanding lesions forms circinate and serpiginous figures. The central area of the lesion shows brownish pigmentation and atrophy. The margin is red. The lesions are commonly asymmetrical.

Involution occurs within about six months. Relapses may however prolong the disease indefinitely. Subjective symptoms are absent.

Diagnosis. The condition is ordinarily characteristic but differentiation from simple purpura, angioma serpiginosum, Schamberg's progressive pigmentary disease, dermatitis hemostatica, and the lichenoid dermatitis of Gougerot and Blum may present difficulties. Progressive pigmentary disease usually occurs in males and is characterized by more or less grouped cayenne-pepper-like red dots which appear on the legs and feet in generally increasing number as new lesions develop; the older ones become darker as they involute and leave a light brown pigmentation.

Prognosis. The prognosis is good. Recurrences may occur.

Treatment. Treatment is of no value.

RAYNAUD'S DISEASE

SYNONYMS *Symmetrical gangrene local asphyxia, primary Raynaud's phenomenon.*

Raynaud's disease is characterized by a trophic usually bilateral disturbance of the fingers, toes, and rarely the nose and ears. The involved parts become cold and assume first a white then blue color turning into red, painful areas. Continued interference with the circulation of involved parts may lead to super

is a local hypersensitivity to cold in the digital arteries. A large number of these patients have primary amenorrhoea. Sterility is common.

Prophylaxis Exposure to cold air and cold water must be avoided. The simple expedient of wearing warm gloves, socks, ear muffs, and galoshes in cold



Fig. 497 Raynaud's Disease.

Local gangrene The course of the disease is progressive. Involvement is, however, paroxysmal. Abortive forms occur in which one or several fingers become lead white only on exposure to cold or to cold water. There is a high associated incidence of scleroderma, especially in old age of the fingers.

Incidence The disease may be familial, occurring chiefly among young and middle-aged women (60 to 80 per cent).

Etiology The cause is unknown. Lewis and others have shown that there

weather may frequently prevent attacks. Smoking is discouraged.

Diagnosis This is easily made from the history. The exclusion of other syndromes which may be similar is important. The age and sex of the patient, the marked seasonal variation in symptoms, and the duration are all very helpful in arriving at a diagnosis. Gangrene when present is minimal and is characteristically limited to the superficial layers of the skin. Immersion of the hands in water at 41° F (5° C) for from five to ten minutes may provoke a typical

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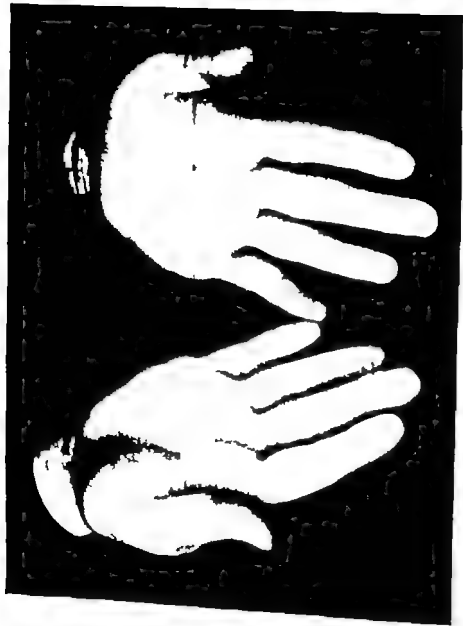


Fig. 199A. Ra nand' syndrome showing extreme pallor and moderate erythema associated with the ascaris produced by exposure to cold. (From *Strand's Diagnosis and Treatment of Carcinoma of the Digestive Tract* Volume II)

seizure. Cervical rib occlusive arterial disease, and organic disease of the nervous system must be excluded.

Treatment. Anemia, hypothyroidism and menstrual disturbances frequently accompany Raynaud's disease and should be adequately treated. In mild cases, this alone may suffice. In the attack the use of *ethyl alcohol* by mouth or

papaverine hydrochloride intravenously 0.033 gm may suffice. The literature is replete with an array of drugs for the treatment of this disease which for some reason give the best results in the hands of the individual authors.

In severe cases, cervical or lumbar sympathetic *ganglionectomy* is the treatment of choice. Some idea of the degree



Fig. 498: Raynaud's Disease.



Fig. 499: Raynaud's Disease.

of relief to be obtained may be predicted by stellate or lumbar sympathetic ganglion block. The use of the drug *tetracyclammonium chloride* (see *Thromboangitis Obliterans*, p. 739) is a simpler

method which will give comparable improvement. The degree of disability produced by the disease determines whether medical or surgical treatment is indicated.

RHINOSCLEROMA

Rhinoscleroma is a chronic infectious disease involving the nose and upper respiratory tract, characterized by scleroma and unsightly deformity.

Incidence. The disease is endemic in South Russia, Austria and Galicia. It

bles the pneumococcus of Friedländer.

Histopathology. The histology is characteristic and consists in a dense plasma-cell infiltration of the dermis, hypertrophy of the collagenous tissue, and the presence of the so-called "Mikro-



Fig. 500 Rhinoscleroma. Involving nose and palate. Note pockered and shrunken mucosa. The sclerotic plaque is stony hard and dusky red in color.

has also occurred in South America. Cases of rhinoscleroma observed in the United States of America have occurred among immigrants from these countries.

Etiology. Rhinoscleroma is probably the result of infection by a short, encapsulated, gram-negative bacillus isolated in 1882 by von Fraenck. The bacillus is readily demonstrable in smears obtained from lesions. The bacillus closely resem-

bles cells. Mikulicz's cells are large, degenerated plasma cells, containing clumps of bacilli of von Fraenck.

Symptoms. The disease begins insidiously as a nodular or diffuse sclerotic enlargement of the nose, palate, and adjoining areas. The nodules are at first hard and freely movable, but fuse to form adherent, immovable, sclerotic plaques. Ulceration rarely develops. Lo-

SARCOMA CUTIS

SYNONYMS *Sarcoma of the skin, sarcomatous cutis, sarcoma cutaneæ, multiple pigmented sarcoma of Kaposi.*

Sarcoma cutis is a malignant connective tissue tumor.

Incidence Scarcely any age is exempt from this affection, although most cases occur either before the age of twenty or over that of forty.

sufficiently pronounced to permit recognition of the original structure from which the sarcoma has arisen, they are classified accordingly into fibrosarcomata, angiosarcomata, etc. If the cells are not so pronounced as to indicate the original



Fig. 503. Multiple Pigmented Hemorrhagic Sarcoma (Kaposi)

Etiology The cause of sarcoma is unknown. These tumors have originated in scars, and the neurofibromas in Reck-Engelmann disease occasionally become sarcomatous.

Varieties The single or localized, nonpigmented type and the multiple, or generalized type comprise the two principal varieties of sarcoma cutis.

Pathology The origin of a sarcoma is sometimes difficult to determine if the cells comprising the tumors are suffi-

ciently pronounced to permit recognition of the original structure from which the sarcoma has arisen, they are classified accordingly into fibrosarcomata, angiosarcomata, etc. If the cells are not so pronounced as to indicate the original

structure, the type of cell determines the name for the tumor, such as spindle cell, round cell, etc. Multiple pigmented sarcoma of Kaposi is a round-cell growth. The pigmentation is due to capillary hemorrhage. The multiple variety may have a structure analogous to a round-cell sarcoma, to a spindle-cell sarcoma, to angiosarcoma, to lymphangoma, or to chronic inflammatory granulation tissue.

Symptoms Nonpigmented sarcoma

sions have a distinct stony hardness are quite insensitive and are of a dark purple or ivory color. Extensive mutilation and deformation occur in more advanced stages of the disease.

Diagnosis Rhinoscleroma is differentiated from *gumma*, *giant-cell sarcoma*, *tuberculosis* and *leprosy* by the presence of the bacilli of von Friesch.

Prognosis Rhinoscleroma is a progressive disease which is very resistant to treatment.

Treatment Roentgen and radium irradiation is the treatment of choice. Improvement has followed autogenous vaccine therapy. Surgery is necessary to relieve obstruction of the nasopharyngeal passages.

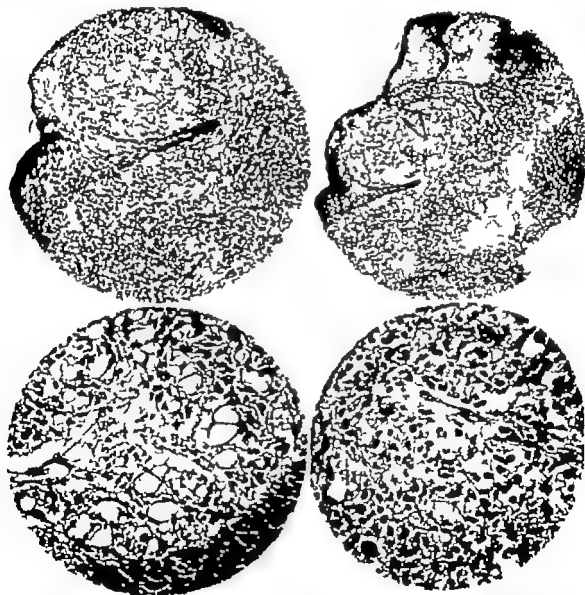


Fig. 501: Rhinoscleroma. Upper Left: Deformity of papillary layer; Mikulicz cells; fibrosis; plasma cells. Upper Right: Deformity of papillary layer; large Mikulicz cells; fibrosis. Lower Left: Mikulicz cells. Lower Right: Plasma cells.

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Symptoms Nonpigmented sarcoma

is characterized by the appearance of one or more small nodules which are reddish blue or flesh-colored. The several lesions increase in size and coalesce to form plaques. Sessile or pedunculated tumors develop on the surface of such a plaque and ulceration may occur. Me-

velop at any age but usually occurs in males between the ages of forty to sixty years. Subjective symptoms are usually absent. It begins on an extremity in the form of firm slightly edematous, diffuse, red spots which sooner or later become infiltrated, red bluish plaques or it be-

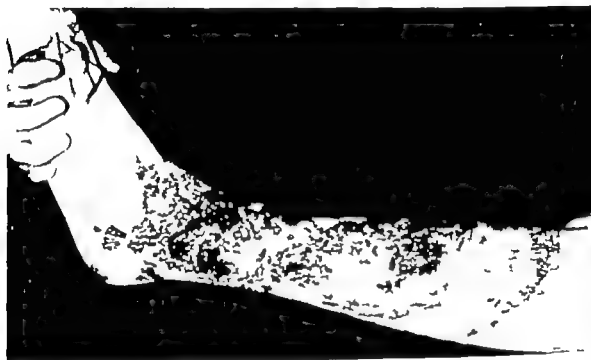


Fig. 503: Multiple Pigmented Hemorrhagic Sarcoma (Kaposi)

tastasis does not occur and the general health is unaffected. Subjective symptoms are absent.

Multiple or generalized sarcoma may be multiple from the start or may follow the appearance of a solitary tumor. The lesions are scattered over the body and may number several hundred. They are flesh-colored or violaceous, and vary in size from a few millimeters to several centimeters. Ulceration commonly occurs. Cutaneous metastasis from a primary sarcoma often occurs. Internal metastasis occurs in from two months to two years.

Multiple Idiopathic Pigmented Hemorrhagic Sarcoma. This may de-

velop as a pea to smaller-sized, bluish-red or even faintly pink, isolated dermic, round or oval, firm nodule on the sole, toes, or palm. If it begins as a plaque, nodules appear in it. If it begins as a nodule, more isolated or grouped nodules appear. They may regress, remain stationary or enlarge and even ulcerate. The vascular character of many of the lesions can be easily demonstrated by pressure which causes a decrease in their size. Those that regress lose their color and their site is replaced by an atrophic scar. Fully developed the clinical picture is that of symmetrical pea to cherry-sized reddish-blue nodules on the hands, forearms, feet, or legs; these nodules are

isolated or grouped into large plaques, the surfaces of which are smooth, irregular or keratotic. Patches in the oral mucosa have been observed. The lymph nodes are not involved until late in the course of the disease. The patient's gen-

dermated area. The nodules grow slowly and as they increase in number and size coalesce to form plaques, the surface of which is flat or marked here and there by variously sized rounded elevations. Here and there, a bluish red nodule may



Fig. 504 Sarcoma. (Courtesy of Dr. Carroll S. Wright.)

eral condition remains good for years. In three to ten years or more, general metastasis occurs and a fatal outcome is usual.

Dermatofibrosarcoma Protuberans. This condition is characterized by closely placed nodules and plaques, occurring anywhere on the body but especially on the trunk. They are painless and firm and appear on palpation to be largely located beneath the skin to which however they are attached. The skin overlying them is either normal or reddish blue in color over part or much of the



Fig. 505 Sarcoma. Inner side of knee.



Fig. 506 Dermatofibrosarcoma Protuberans. Over lumbar area of trunk.

ulcerate and expose a vegetating area. The entire involved area rarely exceeds a diameter of 10 cm. It is an extremely slow-growing localized malignancy with metastases occurring only occasionally and usually after many years. Most histological sections will show the picture of a simple fibroma durum but repeated examinations of different parts of the growth will eventually show in most of the cases the picture of fibrosarcoma.

Diagnosis. Sarcomas must be differentiated from *granuloma fungoides*, leukemia and nodular leprosy. Histolog-

ical examination will reveal the type of tumor.

Prognosis. Generalized sarcoma is always fatal. Fibrosarcoma, if completely removed, does not recur; however it does not of itself produce a fatal termination.

Treatment. Surgery is the treatment for fibrosarcoma. Roentgen irradiation has a palliative effect. For the individual lesions of multiple pigmented hemorrhagic sarcoma, Hollander and Shelton have determined the optimum dose of roentgen rays as 1500 r.

SCABIES

SYNONYMS: *The itch, prairie it h, Cuban it h, swamp it h, Norwegian itch, Philippine itch.*

Scabies is a contagious affection of the skin characterized by multiform lesions and intense itching which occurs chiefly at night.

Varieties. Two forms of scabies are recognized.

1. A variety of the genus *Acarus* infests animals (dogs, cats, horses, and poultry) which man contacts. Animal scabies occurs occasionally in man contacting infested animals. The animal scabies communicated to man is milder than human scabies. Burrows are usually absent in man infested with animal scabies and the lesions are similar to urticaria.

2. Norwegian scabies is a severe form of the disease accompanied by pustulation and crusting. Infestation occurs among individuals living in unsanitary surroundings and those suffering from debilitating diseases, such as leprosy and tuberculosis. The lesions of Norwegian scabies also occur on the face and scalp.

Incidence. It occurs about equally in both sexes of children but, in adults,

males are more frequently affected. It occurs at all ages.

Etiology. The disease is produced by the *Sarcoptes scabiei* which belongs to the Arachnida class. The *Sarcoptes scabiei* found in man does not occur in animals. There are male and female *Sarcoptes scabiei*. The female parasite is gray in color and is barely visible to the naked eye. The male is still smaller and not visible to the naked eye. Each impregnated female mite burrows under the epidermis, where it lives for several months. A female mite is able to deposit on the floor of the cuniculus from one to two ova each day. Ova hatch in from three to six days and reach the adult stage in about a month. Unlike the female the male mite does not burrow or form a cuniculus and can only be identified on the surface of the skin by microscopic examination. A cuniculus appears as a linear elevation of the skin ranging in length from $\frac{1}{4}$ to $\frac{1}{2}$ inch. The female mite inhabits the distal end of a cuniculus. The proximal end is marked by a vesicle, a papul or a

pustule. The female is on the skin surface only at night at which time it feeds and breeds. It is contagious and usually transmitted directly or occasionally through contacts, such as bedclothing. Mellanby in two of 63 trials, transmitted the mite through infested clothing and bedding under most favorable conditions.



FIG. 507 Scabies. Showing sites of predilection, duration two months.

veicle. Ova and feces are present in the burrow and distally the female is found.

Symptoms The presence of burrows or excoriations is pathognomonic of scabies. Papules, vesicles, and pustules are usually present. Secondary infection through scratching masks these symptoms.

The sites of predilection are the flexor surfaces of the wrists, the web and sides of fingers and toes, the ulnar border of the hands, the anterior axillary folds, the ankles, the dorsum of the foot, the umbilicus, infragluteal folds, and the mammae of females. The palms, soles, and



FIG. 508 Scabies. With some unruptured pustules due to secondary infection and with some crusting remains of former pustules. (Courtesy of Dr. Jacques P. Gaequlere.)

Pathology Vertical sections of a burrow show it to be limited to the horny skin layer rarely penetrating the granular layer. The epidermis around the burrow is inflamed and may form a

elbow of infants are frequently involved.

The face is not involved in scabies except among breast-fed infants or those having the Norwegian type.

Itching is rarely present in daytime but intensely aggravated at night.

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a week without the risk of dermatitis and intoxication. It also controls itching and neutralizes the irritating qualities of sulfur.

Cylin. (British Proprietary)	7.5
Ling. sulfur q.s. ad	100.0

The patient is bathed in hot water rich in soap, and the crusts and scales are removed by scrubbing with a soft nailbrush to expose the orifices of burrows. The ointment is then applied to the body surface each night. The body is thoroughly cleansed in a warm soapy bath the seventh morning from the beginning of treatment.

The sulfur content in ointments depends upon the patient's tolerance. A sulfur content of 5 per cent is advisable for older children, women with delicate fair skin, and individuals whose skin is already irritated. Adults with coarse dark skin can tolerate an ointment with from 10 to 15 per cent precipitated sulfur. *Lanolin* and equal parts of *lanolin* and *petrolatum* are recommended as a base during the cold weather. During sulfur therapy dust the skin, after the ointment has been applied, with perfumed talc for its odor and to keep salve from rubbing off.

Continued itching after sulfur therapy commonly means sulfur irritation and a soothing ointment or lotion is indicated.

The following treatments are equally beneficial.

Mitegal (liquid dimethyldiphenylene disulfide) is incorporated in an oily lotion or in an ointment base. A single application is often sufficient to obtain recovery.

Pyralis liquid	0.15
Oil olive	20.00
Spts. rose rect.	20.00

Styrax is useful for young children because it is less irritating than sulfur. It is, however, slightly toxic.

A clean and effective treatment consists in the following. Direct the patient to bathe in warm soapy water dry him self thoroughly and apply a 40 per cent solution of sodium thiosulfate. Wait fifteen minutes, then apply a 4 per cent aqueous solution of hydrochloric acid and wait one hour before a final rinse. Complete recovery follows after several days of treatment.

A single application of colloidal sulfur often effects a cure.

Balsam of Peru is indicated for use as an adjuvant to treatment whenever the skin becomes eczematoid and lichenoid.

Sulfur baths are cleansing but are not effective in treating scabies.

The application of 5 per cent solution of rotenone in quince seed oil has recently received favorable comment.

Sulfur preparations are being rapidly replaced by benzyl benzoate which provides more satisfactory results than any other scabicide and is also the main ingredient of balsam of Peru. Like the sulfur compounds, benzyl benzoate may cause minor or even severe irritation and if it does, treatment should be discontinued.

Benzyl benzoate emulsion (25 per cent) is effective as follows. The body is covered with soft soap, which is worked into a lather with warm water. The entire skin is scrubbed especially the involved areas, with a soft nail brush. The patient then takes a hot bath for ten minutes, following which the benzyl benzoate emulsion is vigorously scrubbed into the wet skin for five minutes. The emulsion should penetrate under and around the fingernails. The skin is allowed to dry and the emulsion is applied again for five minutes. A cleansing bath is taken twenty-four hours later and the patient puts on clean clothes and changes all bed

Diagnosis The diagnosis is based upon the following (1) a study of the sites of predilection (2) by finding burrows (3) by extracting the mite from a burrow or by demonstrating the ova and excrement of the *Acarus* in the burrow. A suspected burrow may be isolated by painting it with writing ink. A preferable

faces of the limbs and areas contacting closely with clothing such as the shoulder, buttocks, and waistline. The seams of clothing invariably reveal pediculi.

Prognosis The prognosis is favorable when adequate treatment is followed.

Prophylaxis This consists of adequate infection from direct scratching

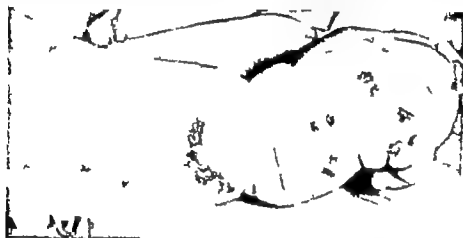


FIG. 509: Scabies. Showing secondarily infected lesion on wrist and hand.

method (Hirschfeld) and a burrow need not be isolated to use it, is to paint the areas of predilection with fluorescein (fluorescein 0.05 gm., glycerin and absolute alcohol, in 200 cc) then clean the painted area with water and examine with a Wood light. The burrow appears as a delicate luminous streak. Any minute fissures on the skin surface which have retained fluorescein will fluoresce greenish yellow while the burrow gives off a grayish white tinge. *Lichen urticatus* of infants and young children is differentiated from scabies by the limitation of lesions to extensor surfaces of the extremities. The lesions of lichen urticatus are papular and may appear as wheals but are free from burrows.

The itching of *pediculosis corporis* is more intense during the waking hours. Lesions of *pediculosis corporis* are characterized by urticarial wheals and see. They commonly occur on extensor sur-

faces of the limbs and areas contacting closely with clothing and bed linen.

Treatment Treatment consists in the destruction of the offending parasite without disturbing the skin and without endangering the patient by possible absorption from medication.

Sulfur ointment is still the classical remedy. It is, however, less effective and more irritating to the skin than *styrax balsam of Peru with nivalal*, or *pyrethrum with betanaphthol*. The following formula suggested by Herbert Smith gives excellent results.

Bal. Peru	15
B naphtholia.	6.0
Sulfur ppt.	10.0
Adeps lana hydroal	10.0
Petrolat. albae	90.0

M. Should be half strength for children.

Locally after hot bath for six nights.

Whitfield lauds the following preparation because it eliminates scabies within

a week without the risk of dermatitis and intoxication. It also controls itching and neutralizes the irritating qualities of sulfur.

Cyflin (Berthak Proprietary)	7.5
Ung. sulfur q.s. ad	100.0

The patient is bathed in hot water rich in soap suds, and the crusts and scales are removed by scrubbing with a soft nailbrush to expose the orifices of burrows. The ointment is then applied to the body surface each night. The body is thoroughly cleansed in a warm soapy bath the seventh morning from the beginning of treatment.

The sulfur content of ointments depends upon the patient's tolerance. A sulfur content of 5 per cent is advisable for older children, women with delicate fair skin and individuals whose skin is already irritated. Adults with coarse dark skin can tolerate an ointment with from 10 to 15 per cent precipitated sulfur. Lascolin and equal parts of lascolin and petrolatum are recommended as a base during the cold weather. During sulfur therapy dust the skin, after the ointment has been applied, with perfume talc for its odor and to keep salve from rubbing off.

Continued itching after sulfur therapy commonly means sulfur irritation and a soothing ointment or lotion is indicated.

The following treatments are equally beneficial.

Mithol (liquid dimethyldiphenylene disulfide) is incorporated in an oily lotion or in an ointment base. A single application is often sufficient to obtain recovery.

Styracis Liquid	0.14
Ol. olive	20.00
Aqta. viad rect	80.00

Styrax is useful for young children because it is less irritating than sulfur. It is, however, slightly toxic.

A clean and effective treatment consists in the following: Direct the patient to bathe in warm soapy water, dry him self thoroughly and apply a 40 per cent solution of sodium thiosulfate. Wait fifteen minutes, then apply a 4 per cent aqueous solution of hydrochloric acid and wait one hour before a final rinse. Complete recovery follows after several days of treatment.

A single application of colloidal sulfur often effects a cure.

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Sulfur baths are cleansing but are not effective in treating scabies.

The application of 5 per cent solution of rotenone in quince seed oil has recently received favorable comment.

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faces of the limbs and areas contacting closely with clothing such as the shoulder, buttocks, and waistline. The seams of clothing invariably reveal pediculi.

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Prophylaxis This consists of adequate secondary infection from direct scratching



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B naphtholis	4.0
Sulfur ppt.	16.0
Adeps lauræ hydrol	16.0
Petrolat. albae	90.0

M Should be half strength for children.
Locally after hot bath for six nights.

Whitfield lauds the following preparation because it eliminates scabies within

na, endocrinopathy, endarteritis, and to primary connective-tissue hyperplasia in the skin. Slight irritation resulting from trauma by a collar-stud, garters, or corsets can be the exciting cause.



Fig. 510 Scleroderma. Note ivory like firmly adherent skin. A case of unilateral scleroderma.

Pathology Scleroderma consists of fibrous-tissue hyperplasia of the dermis and subcutaneous tissue and replacement of fat by fibrous tissue.

Generalized Scleroderma Generalized scleroderma may involve the whole cutaneous surface of the body. The condition is more frequently confined to the arm, leg, and sides of the neck. It may be divided into two stages: a stage of edema and infiltration and a stage of atrophy. Disturbances of the general health in the form of fever, joint pains, neuralgia, and itching may precede or accompany outbreaks. Stiffness of involved parts may be the first and only symptom. This may spread until joint ankylosis occurs, followed by muscular

atrophy. Breathing may become difficult from fixation of the skin and muscles of the chest. Ingestion of solid food may be prevented by mucous membrane involvement.

The skin appears swollen, glossy and hard. The deeper structures become fixed and the natural furrows in the skin disappear. The color of the skin may appear normal or the skin may assume a waxy appearance. Lesions are usually symmetrical.

The disease may begin with the hands. The skin is drawn tightly over the fingers, the joints become fixed, atrophy supervenes, and the fingers tend to become pointed.



Fig. 511 Morphea. (Courtesy of Dr. Jacques P. Guexalerc.)

Morphea Scleriosis (Circumscribed Scleroderma) Morphea scleriosis appears as an oval, definitely indurated patch on the chest, abdomen or extremity. The lesion is ivory in color and surrounded by a lilac tinted zone due to dilated capillaries. It may be aptly likened

linen Humphrey paints the body from the neck down with 33 per cent benzyl benzoate solution in isopropyl alcohol on

two successive days, after which clean clothing is put on, bed sheets changed, and blankets aired

SCLEREDEMA ADULTORUM (BUSCHKE)

SYNONYMS: *Sclerini* (Piffard) *scleredema*.

This is a self limited affection characterized by the sudden onset of a progressive solid edema commonly involving the neck and trunk.

Etiology The cause is unknown. The condition occurs in children and young adults. It has followed a variety of infectious states, among which are tonsillitis, influenza, measles, scarlet fever and mumps and has been seen to develop following the shock of a plunge into cold water. The chief histologic changes are in the presence of swollen connective tissue fibers of the derm.

Symptoms The cutaneous symptoms develop suddenly, sometimes preceded by slight fever and malaise. These subjective phenomena are generally absent when the disease is fully established. At this stage the only subjective sensation is a feeling of being encased in an elastic covering which impedes movement slightly. The disease begins in the integument of the neck and from here advances to the shoulders and usually the entire chest wall. The whole or upper part of the trunk may become involved in a few days or a few weeks. The face and the extremities may or

may not become involved, but the hands and feet are spared. The entire skin becomes hard and inelastic, and cannot be lifted or picked up in folds. Palpation gives the impression of a solid edema involving the deep skin and subcutaneum. There is no pitting on pressure and there are no changes in the epidermis, its sensation, or its color. There are no clinical signs of inflammation.

Diagnosis As Piffard originally pointed out it is ordinarily not difficult to distinguish scleredema from scleroderma even in its early stages. Scleredema develops acutely and invades the neighboring integument rapidly. The epidermis is unaffected and there is often a history of a preceding infection. The hands and feet are unaffected, and finally scleredema has a tendency to spontaneous involution.

Prognosis Spontaneous recovery without sequelae in three to six months is the rule. Recurrences over a period of years have been observed.

Treatment There is no specific treatment. *Physiotherapy* in the form of heat, warm baths, and massage is helpful.

SCLERODERMA

SYNONYMS: *Sclerema adultorum*, *morpheoa*, *dermatosclerosis*, *hidebound disease*, *chelonitis*.

Scleroderma is a chronic disease characterized by hardness and rigidity of the skin.

Varieties It appears in three forms, i. e. generalized, circumscribed (mor-

phoea) and guttate (white spot disease).

Etiology The cause remains obscure. The disease occurs in young adults and more frequently among females. Scleroderma has been ascribed to trophoneuro-

sis, endocrinopathy, endarteritis, and to primary connective-tissue hyperplasia in the skin. Slight irritation resulting from trauma by a collar-stud, garters, or corsets can be the exciting cause.



Fig. 510 Scleroderma. Note ivory like firmly adherent skin. A case of universal scleroderma.

Pathology Scleroderma consists of fibrous-tissue hyperplasia of the dermis and subcutaneous tissue and replacement of fat by fibrous tissue.

Generalized Scleroderma Generalized scleroderma may involve the whole cutaneous surface of the body. The condition is more frequently confined to the arm, leg, and sides of the neck. It may be divided into two stages: a stage of edema and infiltration, and a stage of atrophy. Disturbances of the general health in the form of fever, joint pains, neuralgia, and itching may precede or accompany outbreaks. Stiffness of involved parts may be the first and only symptom. This may spread until joint ankylosis occurs, followed by muscular

atrophy. Breathing may become difficult from fixation of the skin and muscles of the chest. Ingestion of solid food may be prevented by mucous membrane involvement.

The skin appears swollen, glossy, and hard. The deeper structures become fixed and the natural furrows in the skin disappear. The color of the skin may appear normal, or the skin may assume a waxy appearance. Lesions are usually symmetrical.

The disease may begin with the hands. The skin is drawn tightly over the fingers, the joints become fixed, atrophy supervenes, and the fingers tend to become pointed.



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to a piece of celluloid imbedded in the skin. Circumscribed scleroderma also tends to appear in the form of indurated bands of considerable length often on the limbs of children. The lilac border may be absent or present in banded circumscribed scleroderma. As healing occurs the affected areas are replaced by pigmentation of varying duration.

tion are the anterior and posterior aspects of the neck, and the upper part of the trunk. "White spot disease" is not unlike lichen sclerosis et atrophicus (Hallopeau) in appearance.

Sclerodactylia Sclerodactylia, or acrosclerosis, is scleroderma in which the disease extends from forearms to back of hands and fingers. The dorsum



Fig. 512 *Left*: Scleroderma. In a patient with acrosclerosis. Note the sclerosis of the forehead with decreased wrinkling on pressure. *Right*: Acrosclerosis. Note decreased wrinkling over dorsum. Later the skin over the fingers and hands becomes tense and smooth and the terminal phalanges shortened. (Courtesy of Dr. Paul O'Leary)

Guttate Scleroderma (White Spot Disease) Guttate scleroderma is characterized by macular lesions measuring from 0.5 to 2 or 3 millimeters in diameter. Lesions appear as slightly depressed grayish white mother of pearl sheen patches free from the normal lines of the skin. The skin surrounding lesions may be erythematous, violaceous, and hyperpigmented. The sites of predilec-

of the feet and toes may be similarly involved.

Lesions are symmetrical and preceded by vasomotor symptoms consisting of local asphyxia and acrocyanosis, suggesting Raynaud's disease. Tissue contraction occurs without the intervention of infiltration and edema. Fixation of joints, onychogryphosis, and secondary ulcerations occur. Bone absorption com-

plicates the picture. Subjective symptoms and trophic disturbances are absent.

Diagnosis Diffuse scleroderma is not to be confused with *myxedema* in which the parts are softer and not atrophic. Sclerodactylia can be confused with leprosy, syringomyelia, and Raynaud's disease. Sensory disturbances and other cutaneous lesions accompany leprosy. Pain, loss of muscular power and anesthesia are the diagnostic signs of *syringomyelia*.

Raynaud's disease in which acrosclerosis may be present, is characterized by symmetrical atrophy followed by gangrene of the skin and underlying tissues. In infants, subcutaneous induration due to scleroderma must be differentiated from *sclerema neonatorum*, *sclerema oedematosum* and *lipophago granuloma*.

Prognosis Interference with joint movement in diffuse scleroderma leads to serious restriction of the patient's activity. Many of these patients die of exhaustion, uremia, tuberculous, and pneumonia. The prognosis is more favorable for patients with acrosclerosis.

Treatment The treatment of scleroderma is disappointing but spontaneous recovery is not uncommon among children.

Sellei and others champion *pancreatic ferment* given in any of the following forms: (1) raw pancreas in daily quantities of 100 to 200 gm. (3½ ounces) (2) tablets of pancreatic ferments containing amylase, lipase, and trypsin one hour before meals; (3) tablets of duodenal ferments together with pancreas,

and (4) tablets and injections of liver extracts. Continuous treatment over several months is necessary.

Large doses of *viosterol* and *parathyroidectomy* have been recommended. *Sodium thiosulfate* is given intravenously whenever signs of lead and arsenic intoxication are present. Using *dihydrotachysterol* (A. T. 10) 15 drops daily for eighteen days and a rest period of six weeks, Bernstein and Goldberger report a dramatic response in a case of diffuse scleroderma developing one year after a subtotal thyroidectomy. Roop had a similar experience.

Pilocarpine, the salicylates, ammonium chloride, arsenic, iodine cod liver oil, and acetylcholine have been advocated. Hot baths, followed by massage with cottonseed oil containing 2 per cent menthol, are recommended.

The author has obtained favorable results from administering *ammonium chloride* (1 gm. three times daily in a simple elixir) and the consumption of large quantities of *table salt*.

Improvement, in circumscribed forms, sometimes follows intramuscular injections of *barium salicylate* (100 mg. weekly) over a period of three to six months.

In acrosclerosis, O'Leary states that intravenous injections of *fore gm protein*, usually typhoid vaccine have been beneficial in some cases, as has *prostigmine* given orally as the bromide or subcutaneously as the methyl sulfate. In these patients, tobacco should be interdicted and the affected parts kept constantly warm.

SEBACEOUS CYST

SYNONYMS: *Steatoma*, *lipoma*, *atheroma*, *sebaceous tumor*, *steatoma*, *grainsteatoma*

A sebaceous cyst is a painless, encapsulated variously sized rounded and cystic newgrowth situated in the skin or subcutaneous tissue.

Etiology Sebaceous cysts are due to occlusion of sebaceous gland ducts.

Pathology The contents of sebaceous cysts are opaque and of oily consistency. They consist of epithelial cells and their products of degeneration which include fat, fatty acids, cholesterol, saponaceous bodies and occasional calcareous deposits. The capsule is of fibrous connective tissue.

Symptoms The scalp, ears, face, back, and scrotum are favorite sites for sebaceous cysts. Sebaceous cysts are slow-growing neoplasms, taking months or years to reach maturity. They vary in size from a pea to an orange. The skin overlying one of these cysts may be normal in color or may appear pale from distention. The orifice of a gland duct may or may not be present. It gives the appearance of umbilication when the gland orifice is present. The cyst appears as a well rounded projection from the normal plane of the skin when the duct is obliterated.

Sebaceous cysts continue throughout life without giving rise to any symptoms or discomfort. *Steatoma multiplex* is the term used when an individual presents many sebaceous cysts. This is usually observed on the face and on the scrotum.

Diagnosis Sebaceous cysts are distinguished from fatty tumors, gummata, and osteomata. Fatty tumors having a characteristic "pillowy" feeling which is diagnostic. Gummata are usually painful and tender and respond to antituberculous treatment. Osteomata are solid and immovable.

Prognosis Sebaceous cysts are benign newgrowths, showing no tendency to spontaneous disappearance.

Treatment *Surgery* is the treatment of choice. An incision is made over the growth and the entire mass, including capsule, is removed by blunt dissection. This is the usual method but in many cases, especially if the cyst is freely movable, a button of skin and sac roof can be removed under local anesthesia, with a cutaneous trephine (3 to 6 mm. in diameter). Through this opening the rest of the sac may be expressed, sometimes with the help of scissors and forceps. The wound is then dusted with thymol iodide and a bandage is applied with some pressure. Healing usually occurs rapidly and with less scar (Kromayer). Recurrence occurs invariably when the capsule is not completely removed. Small cysts can be incised and their contents expressed. The sac is then swabbed with iodine, silver nitrate or phenol and alcohol.

Electrocoagulation applied to the inner surface of the sac at various intervals produces satisfactory results.

SEBORRHEA

SYNONYMS: *Seborrhea*.

Seborrhea is a functional disturbance of the sebaceous glands characterized by their overactivity and the increased secretion of sebum.

Varieties Two forms of seborrhea are recognized: *seborrhea sicca* and *seborrhea oleosa*.

Seborrhea Sicca. This term identifies

the so-called *vernix caseosa* commonly seen among newborn infants. The condition is a natural process, involving the scalp of infants and lasts for many weeks or months. The accompanying irritation from the decomposing sebum may produce dermatitis. The malady is popularly known among adults as "dandruff." Fatty secretions and exfoliating scales of the epidermis accumulate to form a waxy looking brown coating on the scalp. Seborrhea varies in degree by being slight or so abundant that scales are shed over the shoulders. Mild itching is present. Scaly seborrhea of the forehead is known as *corona seborrhoica*.

A similar noninflammatory condition occurring on the chest is known as "seborrhea corporis." The condition appears in gyrate or circinate formation about the sternum and interscapular regions.

A crusted, scaly seborrhea occurs occasionally on the genitalia of males and females. The pubic region may be involved in the same process.

Seborrhea Oleosa. This is characterized by abundant oily sebaceous secretion. It is commonly seen on the scalp and face. Itching is rarely present in seborrhea oleosa. A tendency to hair loss is present.

The common sites of oiliness on the face are about the nose and forehead. The skin becomes shiny and glistening, and bears a peculiar leaden hue. The sebaceous ducts are patulous. The nose may occasionally be congested and acne rosacea may be evident.

Comedones and *acne vulgaris* may be present in some cases. Seborrhea oleosa is often associated in women with hypertrichosis.

Incidence. Seborrhea occurs among newborn infants and in children between the ages of twelve and twenty years, a period in which the endocrine glands are

most active. It is also common among females approaching the menopause. The disease is universal, has no respect for age or sex, and occurs with greater incidence among males.

Etiology. Malassez ascribed this overactivity to fungus infection. This view has been championed by Unna, Hodara, and Sabouraud.

Anemia, diabetes, nephritis, constipation, and endocrine dysfunction are among the contributory causes. Heredity appears to play a role in overactivity of the sebaceous glands. An oily skin is often a familial peculiarity.

Pathology. Sabouraud believes that seborrhea is due to an overproduction of normal sebum and to dilatation of the duct openings of the sebaceous glands.

Sebum is an oily secretion, and consists of 88.6 per cent mineral fat, 7.2 per cent cholesterol, and 4.2 per cent cholesterol esters, soaps, and phosphates.

Macleod and Dowling have obtained cultures of *M. albida* in scaly seborrhea. The spores of *Malassezia* and colonies of *Staphylococcus epidermidis albus* have been demonstrated in seborrhea oleosa.

Diagnosis. Oily seborrhea is easily recognized by the oily nature of the skin and the regional distribution of the malady.

Seborrhea *seca* should not be confused with *eczema seborrhoicum dermatitis prurians* and *triaz*. The absence of inflammatory signs, the presence of dandruff and the dilated sebaceous ducts are outstanding symptoms in seborrhea *seca*.

Treatment. Internal medication is rarely necessary in treating seborrhea. In extensive cases, large doses of riboflavin and nicotinic acid are useful. The intake of carbohydrates, including sugar, is reduced to a minimum and alcoholic

drinks are prohibited in seborrhea sicca. Acidosis is corrected by the intake of sufficient *alkali* to produce a slightly alkaline reaction in the urine.

Only secretions of seborrhea oleosa are controlled by avoiding fats, carbohydrates chocolate and milk in the diet.

Sulfur in the form of calcium sulfide given in doses of 0.016 gm ($\frac{1}{4}$ grain) or *ichthyol* in doses of 0.2 gm (3 grains) is beneficial.

Temporary relief is attained by *irradiation* with small doses of x rays.

When the scalp is oily daily *shampoos* in the following spirituous soap is recommended.

Thymolis	0.6
Spir. vin. recti	30.0
Saponi molles	60.0

Use After rinsing wet drying scalp, following lotion is rubbed into hair before brushing.

Acid salicylic	2.00
Hydrarg. chlor. corros.	0.15
Spir. vini recti	100.00

Such soap substitutes as acidolate may also be used for the shampoo. The shampoo material should be applied with a toothbrush (moderately soft bristles) directly to the scalp.

The following *colloidal sulfur preparation* and *sulfur camphor lotion* are beneficial in oily seborrhea.

Sulfur (colloidal)	4.0
A. theria	1.0
Spir. vi. recti	1.0
Mucilag. tragacanthae	1.0
Liq. calceae	15.0
Aqua roseae q. d.	30.0

Sulfur is partly soluble in camphor and oil of sesame. It is employed in the following preparation as a lotion.

Sulfur (colloidal)	2.0
Camphorae.	6.0
Oleum sesami	75.0

Use Apply daily with massage.

A synthetic soluble sulfur preparation, known as "thiol" is very useful. It is prepared in liquid and powder forms. The liquid is applied without dilution when a varnishlike covering is formed. The dry powder is used as a dusting powder or may be incorporated in an ointment.

The following additional preparations are effective in seborrhea corporis:

I	
Liq. carbonis detergens	2.0
Sulfur colloidal	1.0
Camphor	0.5
Petrolat	38.0
Sig. Apply t. ice each day	

II	
Mentholi	0.15
Ichth. volis	4.00
Lotio calamineae q. ad	240.00
Sig. Apply daily	

Seborrhea Vasi Seborrhea nasi is treated by *washing frequently* in soap and water and applying a dusting powder containing 30 per cent colloidal sulfur.

Seborrhea of Eyelids Seborrhea of the eyelids is treated with *boric acid ointment* or by 1 per cent *yellow mercuric oxide ointment* or an ointment containing *phenylmercuric nitrate* 1:5000. *Wet dressings* of boric acid or Burow's solution (1:20) are applied to the eyelids several hours each day when acutely inflamed. The following ointment is beneficial in conjunction with wet compresses.

Sol. tione aluminum acetat. (1 per cent)	1.4
Ung. camphor	4.0
Petrolatum q.s. ad	30.0
Sig. Gently massage eyelids.	

Genital Seborrhea. Seborrhea of the glans penis and vulva is treated by *washing frequently* in *boric acid resorcin lotion* a saturated solution of *tannic*

acid or 0.5 per cent aqueous solution of zinc sulfate

Sborrhoea of Scalp The following prescriptions are of value in treating seborrhea of the scalp

I	
Sulfur precip	1.0
Acid salicylic	1.0
Ung. rosae or petrolat	30.0
Rec. Apply once a day (indicated in cases of dry seborrhoea).	

II	
Oxy quinine sulfate	0.15
Menthol	0.50
Acid salicylic	30.00
Liq. carbonis detergens	0.50
Glycerol	2.00
Spir. vin recti (85 per cent)	100.00
Rec. Apply twice each day with massage (indicated in cases of oily seborrhoea)	

III	
Hydroxyethyl chloral hydrate	0.15
Resorcinol	2.00
Acid salicylic	4.00
Oil myrtol	2.00
Spiritus myrtol	5.00
Spir. vin recti (85 per cent)	100.00
Rec. Apply daily to scalp with dropper and massage (indicated in dry seborrhoea)	

Euxoal (monoacetate of resorcin) is substituted for resorcin for individuals with light hair because resorcin darkens the hair

Seborrheal diseases

Oil cadal deodor	10.0
Mineral tarpeth	1.5
Vaselineol	20.0
Oil verbenae q. s. perfume	

Rec. Apply every night and wash scalp following morning (indicated in seborrhoea alopecia).

SPOROTRICHOSIS

Sporotrichosis is a chronic mycotic infectious disease caused by a spore-forming filamentous fungus (*Sporothrix*) which is easily cultured on ordinary media (dextrose agar)

Varieties Sporotrichosis may be classified into two varieties, the cutaneous and the systemic forms.

Incidence It is usually observed in individuals between the ages of thirty and sixty. It is encountered most frequently in gardeners, farmers, vegetable-handlers, and stable groomers.

Etiology The cause of sporotrichosis is a spore-bearing fungus of the genus *Sporothrix*, of which several species have been described in various parts of the world and in all climates. The fungus exists as a saprophyte on vegetables, grasses, and various insects such as fleas and caterpillars. In human beings, the infection occurs through minor injuries of the skin. Vegetables contaminated with the fungus, when eaten raw or only

partly cooked, may infect one through the alimentary canal.

Pathology The tumors of sporotrichosis do not show any specific feature sufficiently characteristic to make an absolute diagnosis histologically. Three zones can be differentiated in these lesions; the outer zone resembles syphilis, the middle zone resembles tuberculosis, while the innermost zone is not unlike that of an ecthyma. On the periphery the connective tissue presents an intense inflammatory reaction. In the middle zone, numerous giant cells of the Langhans type are irregularly distributed. Collections of epithelioid cells similar to that found in tubercles are also present. In the abscessed area, large numbers of pus cells are found lying in a fine or coarse connective tissue network.

It is rare to demonstrate the *sporothrix* in the pus of the lesions. Positive cultures may be obtained by allowing the pus to dry and then planting it

drinks are prohibited in seborrhea sicca. Acidosis is corrected by the intake of sufficient *alkali* to produce a slightly alkaline reaction in the urine.

Oily secretions of seborrhea oleosa are controlled by avoiding fats, carbohydrates, chocolate and milk in the diet.

Sulfur in the form of calcium sulfide given in doses of 0.010 gm ($\frac{1}{4}$ grain) or *ichthyol* in doses of 0.2 gm (3 grains) is beneficial.

Temporary relief is attained by irradiation with small doses of x rays.

When the scalp is oily, daily shampoos in the following spirituous soap is recommended.

Thymol	0.0
Spir. vini recti	30.0
Saponis molles	60.0

Si. After rinsing and drying scalp, following lotion is rubbed into hair before brushing.

Acid. salicylic	2.00
Hydrarg. chlor. corros.	0.15
Spir. vini recti	180.00

Such soap substitutes as acidolates may also be used for the shampoo. The shampoo material should be applied with a toothbrush (moderately soft bristles) directly to the scalp.

The following colloidal sulfur preparation and sulfur camphor lotion are beneficial in oily seborrhea.

Sulfur (colloidal)	4.0
Aetheris	1.0
Spir. vini recti	1.0
Mucilag. tragacanthæ	1.0
Liq. calcis	15.0
Aqua rose q. ad	30.0

Sulfur is partly soluble in camphor and oil of *sassafras*. It is employed in the following preparation as a lotion.

Sulfur (colloidal)	2.0
Camphora	0.0
Oleum sassafr.	75.0

Si. Apply daily with massage.

A synthetic soluble sulfur preparation known as "thiol" is very useful. It is prepared in liquid and powder forms. The liquid is applied without dilution when a varnishlike covering is formed. The dry powder is used as a dusting powder or may be incorporated in an ointment.

The following additional preparations are effective in seborrhea corporis:

I	
Liq. carbonis detergens	2.0
Sulfur colloidal	1.0
Camphor	0.5
Petrolat	30.0
Si. Apply t. i. c. each day	

II	
Menthol	0.15
Ichthyolis	4.00
Loto. calumna q. s. ad	240.00
Si. Apply daily	

Seborrhea Nasi. Seborrhea nasi is treated by washing frequently in soap and water and applying a dusting powder containing 30 per cent colloidal sulfur.

Seborrhea of Eyelids. Seborrhea of the eyelids is treated with boric acid ointment or by 1 per cent yellow mercuric oxide ointment or an ointment containing phenylmercuric nitrate 1:3000. Wet dressings of boric acid or Burow's solution (1:20) are applied to the eyelids several hours each day when acutely inflamed. The following ointment is beneficial in conjunction with wet compresses.

Solutio aluminum cetæ (1 per cent)	14
Ung. aquaphor	4.0
Petrolatum q. s. d	30.0
Si. Gently massage eyelids.	

Genital Seborrhea. Seborrhea of the glans penis and vulva is treated by washing frequently in boric acid resorcin lotion or a saturated solution of tannic

acid or 0.5 per cent aqueous solution of zinc sulfate

Seborrhea of Scalp. The following prescriptions are of value in treating seborrhea of the scalp

I

Sulfur precipip	1.0
Acid salicylic	1.0
Ung rose or petrolat	80.0
Res. Apply once a day (indicated in cases of dry seborrhea).	

II

Oxyquinoline sulfate	6.13
Menthol	0.80
Acid salicylic	30.00
Liq carbolic detergent	6.00
Glycerol	2.80
Spir. lin. recti (83 per cent)	180.00
Res. Apply twice each day with massage (indicated in cases of oily seborrhea).	

III

Hydrogen chloride carbolic	0.13
Resorcinol	2.00
Acid salicylic	4.00
Oil amygdale dulce	8.00
Spiritus myrsine	8.00
Spir. vin. recti (83 per cent)	140.00
Res. Apply daily to scalp with dropper and massage (indicated in dry seborrhea).	

Euresol (monoacetate of resorcin) is substituted for resorcin for individuals with light hair because resorcin darkens the hair

Sabouraud advises

Oil cedar deodor	10.0
Mineral turpeth	1.0
Vanilindol	0.20
Oil ceras q.s. t perfume	
Res. Apply every night and wash scalp following morning (indicated in seborrhea with alopecia)	

SPOROTRICHOSIS

Sporotrichosis is a chronic mycotic infectious disease caused by a spore-forming filamentous fungus (*Sporotrichum*) which is easily cultured on ordinary media (dextrose agar)

Varieties. Sporotrichosis may be classified into two varieties, the cutaneous and the systemic forms.

Incidence. It is usually observed in individuals between the ages of thirty and sixty. It is encountered most frequently in gardeners, farmers, vegetable handlers, and stable groomers.

Etiology. The cause of sporotrichosis is a spore-bearing fungus of the genus *Sporotrichum*, of which several species have been described in various parts of the world and in all climates. The fungus exists as a saprophyte on vegetables, grasses, and various insects such as fleas and caterpillars. In human beings, the infection occurs through minor injuries of the skin. Vegetables contaminated with the fungus, when eaten raw or only

partly cooked, may infect one through the alimentary canal.

Pathology. The tumors of sporotrichosis do not show any specific feature sufficiently characteristic to make an absolute diagnosis histologically. Three zones can be differentiated in these lesions; the outer zone resembles syphilis, the midzone resembles tuberculosis, while the innermost zone is not unlike that of an ecthyma. On the periphery the connective tissue presents an intense inflammatory reaction. In the midzone, numerous giant cells of the Langhans type are irregularly distributed. Collections of epithelioid cells similar to that found in tubercles are also present. In the abscessed area, large numbers of pus cells are found lying in a fine or coarse connective tissue network.

It is rare to demonstrate the sporotrichum in the pus of the lesions. Positive cultures may be obtained by allowing the pus to dry and then planting it

on Sabouraud's medium. Animal inoculation is also successful. In systemic sporotrichosis the blood contains specific immune bodies which show agglutination with a suspension of the spores. In culture media the organism grows as branching septate mycelia with clusters of oval or spherical spores about the ends of the hyphae.



Fig 513: Sporotrichosis. In a farm laborer. Note primary sore and multiple subcutaneous nodules along the lymphatics draining the dorsum of the hand.

Symptoms. The cutaneous variety of the disease as a rule is localized in the skin and the subcutaneous cellular tissue and only exceptionally becomes disseminated. At the site of inoculation a primary sporotrichotic chancre results. In the course of a few days, weeks, or months, an ascending lymphangitis and multiple subcutaneous dusky red indolent painless granulomas develop. The granuloma may soften and form a cold abscess or an ulcer. Occasionally the ulcers are the seat of papillomatous vegetations resembling tuberculosis verrucosa cutis. Sometimes the lesions heal spontaneously but as a rule they persist indefinitely.

The initial lesion is usually located on the exposed part of the body such as the fingers or dorsum of the hand although the leg, face, and other parts of the body may be affected. The period of incubation is from six to twelve days.

The lymphatic type of sporotrichosis is most frequently seen in the Americas. In other countries, especially France, cutaneous sporotrichosis is characterized by multiple disseminated subcutaneous lesions resembling either gumma or tuberculosis, but are polymorphous. Dissemination to other organs is not rare and takes place via the blood stream.

The mucous membranes of the mouth may be primarily involved infection taking place from chewing of infected grains or lettuce.

Symptoms of the Systemic Type. In this variety sporotrichosis may invade the muscles, bones, and joints. The mucous membranes (conjunctivae, mouth, nose, pharynx and intestines) may be secondarily affected. The lungs, kidney and central nervous system are rarely involved. The general health may not be greatly affected although marked cachexia may be present. Subjective symptoms are usually absent.

Diagnosis. The diagnosis is established by the history of the onset, by the indolent character, the linear distribution of the nodules, and by the cultural demonstration of the sporothrix. *Tuberculosis*, *actinomyces*, *blastomycosis*, *syphilis*, *deep trichophytosis* and *streptococcal* infection may resemble

sporotrichous, but are usually differentiated from it without difficulty.

Prognosis. The cutaneous variety of the disease usually undergoes recovery. If the disease is undiagnosed, it may last for years; even then a fatal outcome is rare.

A fatal outcome in systemic sporotrichosis is usually due to tuberculosis or other intercurrent infection.

Treatment. Internal treatment generally results in prompt involution of the lesions. Potassium iodide in doses of 2 to 6 gm. (30 to 90 grains) daily is generally prescribed and should be continued for one month after apparent recovery to prevent recurrences. Intravenous injection of sodium iodide is

equally effective in the dosage of 0.097 gm. (1.5 grains) daily and cautiously. The local areas may be dressed with iodine or tincture or by compresses of Lugol's solution. Mucous-membrane lesions are treated by applications of tincture of iodine.

Sulfathiazole has recently been reported as successful as iodide.

Röntgen ray treatment hastens the resorption of the lesions.

The following is a prescription for iodine ointment.

Iodii puri	04
Butiri lodati	06
Aquaphor	180
Ol. olive	20

See. For local treatment of sporotrichosis.

STRIAE ATROPHICAE

SYNONYMS. *Atrophic lines and spots, atrophoderma striatum et maculatum, atrophica maculae et striae, false cleisterns.*

Striae atrophicae are atrophic striate lesions usually not more than 0.5 cm in breadth and several centimeters in length, somewhat depressed, and usually occurring in parallel lines.

Varieties. There are seven varieties, which are referred to as (1) *linea albicantes*, or *linea gravidarum*, (2) *patellar striae*, (3) *striae of adolescence*, (4) *striae of adiposity*, (5) *striae associated with abnormal tumors or ascites*, (6) *striae of infection*, and (7) *blepharochalasia*.

Incidence. Striae atrophicae occur during adolescence and middle age and are found more frequently in women than in men.

Etiology. The several varieties of striae atrophicae have definite causes. *Linea albicantes* or *linea gravidarum*, are the result of rapid abdominal enlargement. They occur predominantly on the abdomen, but also on the hips and sides of the buttocks. The increase in size of the breasts, either by adipose

tissue or mammary distention due to pregnancy may also produce *linea albicantes* in this region.

Striae occurring over both patellae may follow burnitis and are referred to as *patellar striae*.

The *striae of adolescence* result from rapid growth and are usually seen over the sacral region and anterior lower portion of the thighs. *Striae of adiposity* result from sudden acquisition of adipose tissue. They usually occur on the shoulders, arms, hips, and lateral side of the buttocks. They usually occur in females.

Abdominal tumors and ascites increase intra-abdominal pressure and cause striae in the same location as are noted following pregnancy.

Atrophic striae occasionally follow acute and chronic infectious diseases. The acute diseases causative of striae atrophicae include typhoid fever, dysentery, colitis, appendicitis, sepsis, endocarditis, cerebrospinal meningitis, and

varicella. The chronic infection which may cause atrophic striae is tuberculosis.

Two theories have been advanced for the production of the *striae following infection*. The first theory claims it to be due to the action of toxins on the tissue while the second theory advanced by Finger and Oppenheim regards it as



Fig. 514 Striae Atrophicæ. Atrophic striae involving buttock and lumbar region in a female aged ten years.

due to the rapid increase in adipose tissue which is commonly seen following infections, especially typhoid fever.

The striae of infection occurs most frequently in the lower anterior portion of the thighs, occasionally on the thorax and the arms.

Blepharochalasis occurs on the upper eyelids and is characterized by skin atrophy. The eyelid is dark and wrinkled

Pathology Histological examination shows a rupture of the elastic fibers. The fibers of the cutis have lost their normal structure and all that remain are poorly staining short swollen, and broken fibers. In the striae resulting from chronic illness or malignancy the fatty tissue of the subcutaneous layer is the first to disappear and the skin therefore, lies in folds over atrophic subcutaneous tissue. The atrophy is due to a general malnutrition and eventually all layers of the epithelium are involved. The fibers are much finer nevertheless, they are present while in senile degeneration the elastic and collagenous tissue is destroyed.

Symptoms The symptoms are characterized by the presence of linear streaks of varying length and width occurring on various points of the body as described under Varieties. In color they are purplish grayish or white with usually a glistening or pearly scarlike appearance. They are usually level with the skin, although they occasionally are slightly depressed. They develop insidiously are symmetrical in distribution and are usually multiple.

Pinhead to coin sized spots (maculae atrophicæ) of similar appearance and slightly depressed may be the sole manifestation of this disease or they may be associated with the linear variety. When they occur as the only manifestation of the disease one finds them located on the trunk and extremities.

Subjective symptoms are absent.

Prognosis and Treatment The condition is permanent, and treatment is of no avail.

SUBCUTANEOUS INDURATION IN THE NEWBORN

Howard Fox enumerates four principal types of subcutaneous induration occurring in the newborn or young infants.

I Scleroderma It is exceedingly rare. The clinical diagnosis and differentiation are difficult. The course of the disease is

suggestive and histologic study is conclusive since the dermic lesions are similar to those seen in generalized scleroderma of adults (see p. 870).

II. Sclerema Neonatorum (Sclerema Adiposum) The affection appears suddenly during the first weeks of life in undernourished premature, or debilitated infants. The characteristic lesions consist of very hard, irregular subcutaneous masses the size of a hen's egg. These appear first on the lower extremities, usually the calves, and rapidly extend upward. They may involve the entire surface except the palms, soles, and scrotum. There is no pitting on pressure. The skin has a whitish or waxy color and is cold to the touch. The disease is fatal usually in a few days.

III. Sclerema Oedematosum (Oedema neonatorum) This develops about the third or fourth day of life in weak, premature, atrophic infants. It consists of a pitting edema of varying extent. Usually the dorsa of the feet, lower extremities, eyelids, and upper extremities show involvement. Circumscribed indurations are not present. Death usually occurs within a week.

IV. Subcutaneous Fat Necrosis (Lipophagic Granuloma) This is probably the commonest form of subcutaneous induration observed in infants. The disease occurs in healthy, often large infants and is a self-limited, localized process affecting various sized areas of the

body often in a fairly symmetrical manner. The lesions appear from two to twenty days after birth as deep-seated, indurated areas in the subcutaneous tissue. They vary in size from that of a split pea or a hen's egg to large areas covering the greater part of the back and buttocks. The color of the skin over the hard areas is usually bluish red at the outset. This disappears gradually the skin assuming its normal color even before the lesions have softened and disappeared. The skin surface is smooth, and there is no evidence of pain on pressure. The induration does not pit on firm pressure, and the hardness is often described as resembling that of wood or rubber. The indurated areas may or may not be movable on the deeper parts" (Howard Fox).

According to Lemes, the favorite sites are first the back, then cheeks, arms, thighs, and finally the buttocks in the order given. The palms, soles, and abdomen are not involved.

Trauma is believed to be the basic etiologic factor since these infants are often born after a difficult labor or following overzealous slapping to induce or hasten resuscitation. As a result of the trauma localized necrosis of the subcutaneous fat is produced with the formation of the foreign-body granuloma found histologically. Cure is spontaneous softening and absorption begins about the sixth week and is complete in three to four months.

SUDAMINA

SYNONYMS *Miliaria crystallina*, *sudamina*.

This is an acute, ephemeral, non-inflammatory dermatosis, characterized by an abundant eruption of small thin-walled bullae.

Etiology The condition seems to be due to a temporary functional derange-

ment of the sweat-secreting apparatus. A sudden excessive sweat secretion following temporary anhidrosis appears to lead to a separation of the horny-cell layer with the formation of minute blebs. Prior to this development the

varicella. The chronic infection which may cause atrophic striae is tuberculosis.

Two theories have been advanced for the production of the *striae following infection*. The first theory claims it to be due to the action of toxins on the tissue while the second theory advanced by Finger and Oppenheim regards it as



Fig. 514. Striae Atrophicae. Atrophic striae level ing buttock and lumbar region in a female aged ten years.

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The striae of infection occurs most frequently in the lower anterior portion of the thighs, occasionally on the thorax and the arms.

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Pathology. Histological examination shows a rupture of the elastic fibers. The fibers of the cutis have lost their normal structure and all that remain are poorly staining short swollen and broken fibers. In the striae resulting from chronic illness or malignancy the fatty tissue of the subcutaneous layer is the first to disappear and the skin therefore, lies in folds over atrophic subcutaneous tissue. The atrophy is due to a general malnutrition and eventually all layers of the epithelium are involved. The fibers are much finer nevertheless they are present while in senile degeneration the elastic and collagenous tissue is destroyed.

Symptoms. The symptoms are characterized by the presence of linear streaks of varying length and width occurring on various points of the body as described under Varieties. In color they are purplish grayish or white with usually a glistening or pearly scarlike appearance. They are usually level with the skin, although they occasionally are slightly depressed. They develop insidiously are symmetrical in distribution, and are usually multiple.

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Howard Fox enumerates four principal types of subcutaneous induration occurring in the newborn or young infants.

I Scleroderma. It is exceedingly rare. The clinical diagnosis and differentiation are difficult. The course of the disease is

puberty originate in a hair anlage at puberty and are distributed in certain areas (axilla, genitalia, groin, perianal region, and mammary glands in the female). It is a large compound tubular gland whose secretion consists of partly degenerated cells. According to Homma, 70 per cent of these glands contain iron. These are the glands presumably involved in hidradenitis suppurativa (see p. 410). Some 80 per cent of the unnecessary bodily heat is dissipated through the skin. This is accomplished by a constant, insensible perspiration. There are wide variations in the activity of the sweat glands within physiological limits. A pathological increase of perspiration is termed "hyperidrosis," its absence anidrosis. However these terms generally refer to conditions in which the increase or decrease is persistent or habitual.

Hyperidrosis

Excessive sweating may be localized or generalized. In generalized hyperidrosis, the sweating is apt to be more marked in certain parts of the body especially the forehead, axilla, genitalia and groins, chest, palms, and soles. It occurs at the outset and at the termination of certain febrile infections, in certain cathectic states, and especially in pulmonary tuberculosis. It occurs in hyperthyroidism, in obesity and in gigantism, probably from the accompanying hyperthyroidism. It has been observed in paresis, myelitis, and injury to and tumors of the brain. Miliaria and sudamina often complicate the general type incident to febrile paroxysms. In malaria, the attacks are periodic. Some sweat easier than others. This predisposition is greater in those with an unstable involuntary nervous system in whom it is the result of direct

or reflex action. That following exertion or excessive bodily heat production and psychic influences (fear, nervousness, fatigue and mental work) is well known. Eating hot or spicy foods and sometimes tomatoes, pickles, onions, or chocolate, causes reflex sweating of varying degree especially of the face, in certain persons.

Localized Hyperidrosis. It is this form of hyperidrosis for which the dermatologist is usually consulted. Instances of hyperidrosis limited to small cutaneous areas such as a small area of the forehead, the dorsum of a hand or tip of the nose have been observed. These are rare. It may only involve one side of the face, trunk, or a limb. Hyperidrosis or anidrosis may follow a traumatic nerve lesion or other organic disease of the nervous system but rarely as an isolated symptom. The common forms of localized hyperidrosis involve the axilla, the palms, the soles, and the genitalia. Reflex axillary hyperidrosis is common in persons who undress for a physical examination. Axillary hyperidrosis, with or without odor to the perspiration, is exceedingly common particularly in women. The palms of many people are constantly moist—often worse as a result of the slightest emotion and during the summer. In certain occupations, such as accountancy and bookkeeping, it may become a serious problem. Sweating of the soles is also common. In this, as in sweating of the palms, the parts may be cold, clammy and blue (acrocyanosis) or they may be warm and the skin erythematous. In some the skin may become softened, macerated, whitish, and give off a particularly fetid odor (bromidrosis). This latter condition may however occur without hyperidrosis. The odor is due to the secretion from the apocrine glands or is body odor in general.

sweat duct appears to have been temporarily obstructed resulting in an escape of sweat into the surrounding horny layer. The condition is therefore common in those undergoing febrile bouts and in those seriously debilitated.

Symptoms. The eruption develops suddenly without subjective symptoms, and generally on the anterior part of the trunk and forehead. The lesions appear in a size ranging from a pinpoint to a pinhead and as whitish translucent blebs with exceedingly thin wall since they break on the slightest touch. They are discrete and usually abundant. They coalesce exceptionally to form larger bullae but their contents remain

clear throughout the short course of their existence. Left alone they dry and degenerate, the entire process lasting from several days to a week, although new lesions may appear and prolong the condition as a whole.

Diagnosis. The diagnosis is not difficult. In *vesicular eczema* and *milium* there are always inflammatory phenomena. In *hydrocystoma* there is no general debility and the lesions are firm and deep-seated and limited to the face.

Treatment. The disease is self-limited. It is sufficient and generally refreshing to sponge the body with dilute alcohol and to follow this with a dusting of purified talc.

SWEAT-GLAND DYSFUNCTION

Disturbances in the secretion of sweat may be quantitative (anidrosis and hyperidrosis) or qualitative (bromidrosis, uridrosis, hemidrosis or chromidrosis). Strictly speaking none of these sweat alterations is always the result of dysfunction. They may be normal under certain circumstances, incidental but also normal for the body in the presence of certain general disease states, and finally one or more of them may be truly the result of a dysfunction.

Sweat product of the secretory activity of the coil or sudoriporous glands is a clear watery liquid with a specific gravity of 1.002 to 1.003 and a hydrogen ion concentration according to different observers, of 4.0 to 7.5. The pH value of the average normal skin is about 5.5. Its odor varies with the particular cutaneous area of its origin, the person and the race, as well as food (especially onions and garlic) and drugs ingested. According to Peck and his associates the composition of sweat is as follows:

Water	99.02
Sodium chloride	0.70
Lactic acid	0.10
Acetic acid	0.0006
Propionic acid	0.0003
Caprylic and/or caproic acid	0.0046
Citric acid	0.010
Ascorbic acid	0.004
Urea	Trace
Uric acid	Trace

Sweat glands are under the control of the sympathetic nervous system. Certain drugs (acetylcholine, pilocarpine) stimulate these glands; others (atropine) suppress their secretion. The ordinary or normal stimulus to sweat secretion is a rise in blood temperature which acts by stimulating the nerve centers. Sweat glands have been differentiated by Schiefeldecke as follows:

Eccrine glands originate in epidermis during fetal life, are distributed generally but are especially numerous on the palms and soles, are small simple tubules, and secrete a simple fluid sweat.

Apocrine glands become active about

water and a bland dusting powder should be used. A dusting powder of 3 to 10 per cent *salicylic acid* in talc is often helpful in bromhidrosis.

Thoroughly tanning the affected parts by means of ultraviolet radiations has a distinctly drying effect. *Plastic resection* (in axillary types) may be indicated in some cases of bromhidrosis. *Division of nerve* supplying an affected area may be necessary in some.

Acrocyanosis (Acrosphyxia)

This term is used for a functional circulatory disturbance of the extremities characterized by chronic passive congestion. The hands, and less often the feet, are cold, clammy, perspiring, often slightly swollen, and present varying degrees of a more or less uniform, red-blue color change. The color change is often sharply demarcated above the wrist. In some even the buttocks, ears, and nose may be cold and show this violet red color. It is commoner in women.

There may be no other symptoms and the patient is apparently in good health. In some males, it appears to be associated with effort syndrome. In others it is associated with hypothyroidism, ovarian dysfunction and various neuroses. It has been observed to follow debilitating illnesses and is occasionally seen in chronic arthritis. It is often seen involving the palmed extremity of children who have recovered from infantile paralysis. It is in this condition that it is apt to reach its most marked development and in which nodules suggesting erythema in duration are not uncommon.

According to Lewis, the disturbance is arteriolar with angiospasm and capillary anoxemia. For Layan, it is due to venous capillary stasis with dilatation of the venules. Continued circulatory deficiency may produce nutritional changes in the

affected area. A Telford states, subcutaneous nodules may form which commonly disappear but occasionally break down and lead to a chronic painful ulceration. This is especially observed in cases of old, anterior poliomyelitis.

Clinically acrocyanosis should be distinguished from *cutis marmorata* (*livedo reticularis*) in which the reddish blue color changes appear in the form of a noninfiltrative network particularly on the upper and lower extremities. The round or oval spaces enclosed by the network are normal in color.

Under the term "erythrocyanosis," a closely related condition appearing in stout, florid well nourished girls and



Fig. 515 *Livedo Reticularis*. Unusually marked on forearms and legs.

young women has been described. It consists in a reddish-blue congestive discoloration seen on the lower half of the legs. Cold intensifies the condition. It is particularly marked in the areas above the malleoli whereas in acrocyanosis it

Treatment *Generalized Hyperidrosis*

This is based on the underlying cause however it is difficult even when it is symptomatic. In those in whom no etiologic factor is found *empirical treatment* is suggested. In some instances the patient may have to be hospitalized the water and sodium-chloride intake reduced and the actual amount of perspiration recorded. *Quinine* and *atropine* to the point of tolerance may be beneficial but only temporarily. The following internal remedies have at times been found beneficial. *Barbiturates* or *bromides* to reduce any existing nervous tension and empirically *arsenic* *iron* *ergot* or *enteric-coated pills* containing *camphor* and *sulfur* in teaspoonful doses twice daily. Local applications as indicated below or weak solutions of *alum* or *tannic acid* may help.

Localized Hyperidrosis. In localized hyperidrosis *deodorants* and *antiperspirants* are commonly used together since localized sweating is often accompanied by disagreeable odor. In foot cases socks stockings and shoes should be changed daily. Light weight footwear is advisable. The affected parts should be washed with soap and water several times daily. A *lotion* such as the following may be applied after each washing.

Prepared neocalamine	24
Vedli boriel	40
Acid alkylies	24
Acid benzoxel	48
Sp. vini rect	600
Aq qs d.	1200

A good *deodorant cream* is as follows

Benzole oil	0.6
Zinc oxide	6.0
Aquaphor	32.0
Perfume q.s.	

If this does not produce the desired result *formalin* (1 per cent in water or $\frac{1}{2}$ per cent in 70 per cent alcohol) may be

tried applied twice daily. Soaking the hands or feet in 5 per cent *formaldehyde solution* is helpful but must be used with caution. According to Freis, *iontophoresis* for twenty minutes daily for three days, using 1 per cent formaldehyde solution will check sweating of the hands or feet for three to four weeks. A 0.1 to 1 per cent solution of *potassium permanganate* is also useful. Potassium permanganate is both antiseptic and deodorant. Stains may be removed with vinegar lemon juice or dilute acetic acid. It may be prescribed in 1 to 5-gram tablets or in a 5 per cent concentrated solution which is diluted when used. Goodman recommends the following dusting powders

I	
Zinc peroxide	1.0
Benzoc acid	1.0
Talc q.s.	100.0
II	
Magn carbonate	32.0
Alum	32.0
Talc q.s. ad.	100.0

Aqueous solutions of *aluminum chloride* (20 per cent to 30 per cent) are especially helpful. The solution should be applied every other day allowed to dry and the parts then dusted with talc or bentonite. No soap should be used before its application. It should not be applied to broken skin or used immediately after shaving under the arm. After effective results have been obtained applications are made less frequently. Of the physical agents, *roentgen ray therapy* is the most efficacious, but moderately large doses are required. It should be used only in adults. The dose advised by MacKee is 75 roentgens weekly and two to six months treatment may be necessary. Overdose may lead to persistent dryness which may not appear until years later. During the therapy and for three weeks afterwards, nothing but soap and

common, and toxic agents from focal infections, such as chronic tonsillitis, chronic sinusitis, chronic otitis media and dysentery.

Symptoms The patient often complains of itching or burning. This is worse when marching in heavy footwear. There are objectively according to Park, the following, any one or two of which may be absent.

- (1) Hyperidrosis which may be very severe and fatal. The horny layer may become sodden, whitish, or fluid on the parts of greatest pressure.
- (2) Erythema involving the pressure areas, especially the heel, outer border of sole, anterior arch, and plantar surface of the toes.
- (3) Hyperkeratosis which favors the same areas and projects above the surface of the edges of these areas.

In atypical cases, only erythema and burning are present, hyperidrosis is rarely absent.

Prognosis It is self-limited but recurrences are common. The patches disappear when the excessive sweating is controlled. Park states that rest in bed leads to a rapid return of the soles to normal.

Treatment This consists in the removal of all chronic, septic foci. The routine used by Park and his colleagues consists in soaking the feet for fifteen to twenty minutes, twice daily, in a warm solution of potassium permanganate (1:4000) after the feet are dried, they are powdered with 5 per cent salicylic acid in talc. Milder forms may be relieved by the use of the following creamy lotion.

Methuenine	0.5
Tricresath	0.5
Talc	83.0
Water	75.0

See. Spread over feet and between the toes and allow to dry.

Granulosis Rubra Nasi (Jadassohn)

Definition This is an affection characterized by (a) redness of the front and sides of the nose and sometimes the forehead and chin, (b) the presence of minute, flat, pink or red papules on the erythematous base, (c) constant hyperidrosis of the affected parts. Droplets of sweat are constantly present, and the nose is apt to be cold, damp, and shiny. The papules disappear under glass-pressure, thus differentiating the disease from lupus vulgaris. It is at times mistaken for acne rosacea.

Etiology It is commonly seen in children and tends to disappear little by little spontaneously usually by puberty. It is probably of endocrine origin although the histologic lesions are in the nature of a chronic inflammation involving the peripheral capillaries in the vicinity of the sweat glands.

Treatment Astringent lotions containing two to five per cent acid salicylic or resorcin in witch hazel water are helpful. This condition has been controlled by radiation with x-rays.

Anhidrosis

A temporary or permanent decrease or complete absence of sweating of a localized or generalized type is seen in many chronic dermatoses such as eczema, psoriasis, dermatitis exfoliativa, erythrodermas in general, and in senile skin ichthyosis, and xeroderma pigmentosum. It occurs in minor and major ectodermal defects (see p. 310). It occurs also as a symptom of certain systemic diseases, such as tuberculosis, diabetes mellitus, diarrheal states, cancer, hypothyroidism, and cerebellar states in general. Winter itch, or pruritus hiemalis, is the result of a decrease in sweat and sebaceous-gland se-

■ the areas served by most peripheral parts of the circulation (digits nose or ears) which are chiefly involved

In marked instances, the skin of the lower half of the leg is purplish and cold to the touch the hair follicles appear as

Symmetrical Lividities of the Soles

SYNONYMS: *Symmetrical erythema of the soles, scalded feet.*

Etiology The condition occurs in young adults. It is a common dermatosis



Fig. 516 Symmetric Erythema. Of soles. (Courtesy of Drs. J. M. Hill and Robert F. Hansen.)

small red puncta and there is palpable a firm, elastic, subcutaneous infiltration of varying degree. According to Telford the nodules which appear from time to time in this condition are not tuberculous nor different from those seen in erythema induratum of Bazin (see p 781)

affecting men under military conditions in certain climates. Tate enumerates the following possible etiologic factors: A susceptibility to infective, circulatory, sensory and secretory cutaneous disturbances, psychoneuroses, in which sweating of the hands and feet is not un-

organisms are virulent enough to cause permanent hair-follicle destruction and ectectroea. In such cases, the minute crustacea may appear over large areas or may start in the center of a large plaque and spread centrifugally; the skin becomes smooth and sclerostrophic as a

signs. In some, these signs are exceedingly acute and distressing; in others, especially in a condition of great chronicity these signs are slight in comparison to the patient's appearance. The site of predilection for sycosis vulgaris is the bearded region, but it may involve



Fig. 518 *Left* Sycosis Vulgaris. *Right* After course of injections of staphylococcus filtrate. Staphylococcus vaccine (toxoid or Va-Tox-Malford) is occasionally of value in ectectroic sycosis.

result of destruction of the hair follicles, and there is permanent loss of hair. This variety has been termed "lupoid sycosis" or "ulcerthema sycosiforme," and is seen on the face and occasionally elsewhere. The active border of the process may suggest lupus vulgaris, but typical lesions of that disease are absent and only follicular pustules seen. In some cases of sycosis only a small region may be involved, such as the upper lip or the lower lip below the vermilion border. Subjective sensation of itching, burning pain, tenderness, and cutaneous tension are constant.

The extent of the infection alters the degree of both objective and subjective

the upper lip, eyebrows, scalp, axillae and pubis. Some parts of the affected region may vegetate.

Diagnosis. The diagnosis is generally not difficult. In the deep type of *true* or *parasitic* sycosis there is distinct lumpiness and large cutaneous swellings; the hairs on microscopic examination show the fungi. Sycosis vulgaris may be differentiated from *eczema* by the fact that it is rarely limited to the bearded or mustache region, usually spreads to non-hairy parts, and is not purely a follicular disease; in *eczema* furthermore vascular or crusted and minute eroded regions can be found.

Prognosis. The disease is chronic,

cretious due to wind and weather and is often accompanied by eczematization of the skin

In Horner's syndrome which follows section or paralysis of the cervical sympathetic, there is, besides ptosis, miosis and enophthalmos, vasodilatation, higher local temperature and anhidrosis over the affected side of the face. Irritation of the cer-

vical sympathetics causes opposite effects.

The anhidrosis in those with mild and even severe ichthyosis can and should be alleviated by means of hot baths, Turkish baths in particular.

Hydrocystoma, sudamen miliaria, and hidradenitis suppurativa, all sweat-gland disturbances, are discussed under those headings.

SYCOsis VULGARIS

SYNONYMS: *Folliculitis barbae*, *coccogenic sycosis*, *nonparasitic sycosis*.

This is a chronic suppurative staphylococcal infection of the hair follicle, especially of the bearded and mustache regions, characterized by an inflammatory papulopustule pierced by a hair.

Etiology. Sycosis vulgaris is seen in the male only. It is a primary superficial—sometimes deep—follicular infection by *Staphylococcus aureus*. In sycosis of the upper lip, a dermic nasal discharge may be a predisposing factor and is often the cause of its prolongation. In the bearded region, sycosis vulgaris may be initiated by injury to and secondary infection of one or several hair follicles by a dull razor.

In most cases, the disease appears to require a special type of skin for its development and persistence. Most patients are apparently in good general health. Seborrheic dermatitis may be the basis for an extensive and extremely rebellious suppurative folliculitis of scalp, face, eyebrows, axillae, and pubis.

Symptoms. The onset is gradual. The initial lesion is clinically a perifollicular dull red papule which develops first as a red point. Generally a minute yellow pustule centered by a hair then appears and enlarges for several days. On palpation, one senses the deeper epidermic infiltration. As new lesions appear in the vicinity, a well-defined indurated and

inflammatory area gradually develops. The surface of this plaque is bright red, somewhat swollen, eroded, and crust covered in spots, and is marked by numerous suppurating follicles. If a hair



Fig. 517. Sycosis Vulgaris. Showing widespread, discrete involvement of three years duration.

is withdrawn the root is found to be coated with a whitish translucent gelatinous material. In most instances, the hairs sooner or later come away easily on traction but regrow. Occasionally the

organisms are virulent enough to cause permanent hair-follicle destruction and cicatrices. In such cases, the minute pustules may appear over large areas or may start in the center of a large plaque and spread centrifugally; the skin becomes smooth and sclerotropic as a

signs. In some, these signs are exceedingly acute and distressing; in others, especially in a condition of great chronicity these signs are slight in comparison to the patient's appearance. The site of predilection for sycosis vulgaris is the bearded region but it may involve



Fig. 518 *Left* Sycosis Vulgaris. *Right* After course of 1 injection of staphylococcus serum. *Staphylococcus vaccine* (travail de Va-Tux—M Howard) is occasionally of value in coracogenic sycosis.

result of destruction of the hair follicles, and there is permanent loss of hair. This variety has been termed "lupoid sycosis" or "erythema sycosiforme" and is seen on the face and occasionally elsewhere. The active border of the process may suggest lupus vulgaris, but typical lesions of that disease are absent and only follicular pustules are. In some cases of sycosis, only small region may be involved, such as the upper lip or the lower lip below the vermilion border. Subjective sensations of itching, burning pain, tenderness, and cutaneous tension are constant.

The extent of the infection alters the degree of both objective and subjective

the upper lip, eyebrows, scalp, axillae, and pubes. Some parts of the affected region may vegetate.

Diagnosis. The diagnosis is generally not difficult. In the deep type of *tinea* or *parasitic sycosis* there is distinct lumpiness and large cutaneous swelling; the hairs on microscopic examination show the fungi. Sycosis vulgaris may be differentiated from *eczema* by the fact that it is rarely limited to the bearded or mustache region, usually spreads to non-hairy parts, and is not purely a follicular disease; in *eczema*, furthermore, vesicular or crusted and minute eroded regions can be found.

Prognosis. The disease is chronic,

and shows little tendency to spontaneous cure. Untreated it is apt to persist for years. The earlier treatment is instituted the better. Excellent results can be obtained in the majority of patients.

Treatment. The indications are (1) to remove any crusts containing virulent organisms, which prevent the direct action of lotions and salves; (2) to apply soothing and antiseptic lotions and salves to the inflamed infected skin; (3) to remove the hairs from the infected follicles, without which syosis vulgaris cannot as a rule, exist; (4) to raise the patient's general resistance through proper hygiene, a diet such as is suggested for acne vulgaris, and injections of staphylococci vaccine or toxoid; and (5) to correct contributing factors where possible such as a chronic nasal discharge and blepharitis. In some the disease can be checked quickly, in others, only after prolonged and detailed treatment.

Lotions and Salves. In early cases or in those with small circumscribed areas, manual depilation and the use of any of the lotions or salves noted below may be sufficient to bring about a cure. In all cases with acute manifestations of inflammation (pain, tenderness, burning) only soothing applications should be used for several weeks. The following in the form of wet, cold or hot constant compresses are suggested:

I	
Saturated boric acid solution	
II	
Resorcinol	30.0
Boric acid	30.0
See 1 teaspoonful to pint hot water	
III	
Solution of aluminum acetate	240.0
See 1 to 2 tablespoonful to pint water	
IV	
Ichthyol	150.0
See 3½ teaspoonful to pint of water	
V	
Potassium permanganate 1:1000	

At night a simple powder lotion—calamine lotion with 3 per cent boric acid—may be used. During this period the hair should be clipped short. When the condition becomes less acute or if it is not acute when the patient first comes under observation shaving daily with an electric or safety razor (using a fresh blade each time) is advisable. This procedure however becomes unnecessary if x-ray depilation is performed. An excellent antiseptic lotion used several times daily is 70 per cent alcohol, a 1:1000 aqueous solution of phenol, or the following:

Bichloride of mercury	0.1
Alcohol	100.0
Water q.s. ad	240.0

Possibly as a result of previous treatment the skin of some patients appears to be extremely irritable and sensitive to various applications, making it necessary for the therapist to test his prescriptions on small cutaneous areas. The following in ointment form have been found of value from time to time: calomel (3 to 5 per cent), ammoniated mercury (3 to 8 per cent), yellow oxide of mercury (5 to 10 per cent), ichthyol (5 to 20 per cent), chlorhydrxyquinoline (0.5 per cent), plus benzoyl peroxide (10 per cent) in a greaseless ointment base (Peck) or quinolor (Squibb), sulfur precipitate (5 to 25 per cent) in cholesterolized petrolatum or greaseless ointment base (Burroughs-Wellcome) or even in a lotion is at times of great value. Results with the antibiotics, penicillin in particular topically and by injection, have not been uniformly beneficial.

Removal of Hairs. Manual or x-ray depilation should be practiced whenever possible. In the circumscribed types, manual depilation is easy. In extensive cases it is often too trying on the patient. In these, x-ray depilation is indi-



Fig. 519 Syrach Corrugentia. (Courtesy of Dr Carroll S. Wright.)

cated. Indeed, it represents the most valuable therapeutic measure at present at our disposal. Although fractional doses are used by many (one-fourth erythema dose weekly for from eight to ten exposures) it is the author's opinion that in all extensive cases the sooner depilation is obtained the quicker the results. The depilating dose is best fractionated in one-fourth erythema dose every fifth day for five exposures. No medication should be used for at least one week before the x-ray exposures. In most patients, the hair will loosen and be easily removed in from fourteen to eighteen days after

the last exposure. The antiseptic lotions or medicated salves already enumerated should be used following depilation to prevent relapse so far as possible.

Not all patients require the drastic step of depilation, a certain percentage can be cured by chemotherapeutic agents alone.

In persistently recurrent types, x-ray exposures must be continued until depilation has been permanently obtained before cure is established. In these, effects of the persistent sycois, as well as those attributable to the radiation, must be expected.

SYNOVIAL LESIONS OF THE SKIN

SYNOVIAL *Periarticular cysts.*

These are rare, cystic lesions beneath the skin commonly observed in the vicinity of joints, especially the dorsal aspects of the fingers. They begin as small, later pea-sized, semi-translucent, fluctuating, globular elevations. The lesions are persistent and painless unless their contents produce overdistention. The skin overlying them is thin, shiny, smooth, or verrucous, and a narrow red border is usually seen around the base of the lesion. When punctured, yellowish mucoid, or grumous fluid exudes and continues until the puncture wound heals at which time the cyst reforms. They resemble the ordinary ganglion of tendons because they are close to tendons and contain the same sort of contents.

Mc Kee and Andrews have skiagraphically demonstrated their connection with the adjacent joint cavity and state that a canal-like or fibrous cord connects the capsule of the cyst with the contiguous joint. Hailer, after a study of five cases previously injected with iodized poppy-seed oil, had the impression that they de-

velop from the synovial membrane lining tendon sheaths and are connected with the sheath canals and not with the nearby joint cavities. Montgomery and Culver think they are verruca of a special type because when curetted, they show a palisade structure similar to what is seen when warts are curetted.

They must not be confused with traumatic epithelial cysts (Wein and Caro) which occur on the palmar surfaces and occasionally the plantar surfaces and elsewhere as freely movable, painless, oval, pea-sized, rather firm swellings beneath the skin. In these a scar may be present to indicate the originating trauma.

Treatment. X-ray therapy, radium, electrolysis and careful dissection with resection have been found curative in selected cases. Incomplete removal is followed by recurrence, and radiation is more often disappointing than successful. When radiation is used, maximum, crossfire exposures at monthly intervals are preferable.

SYPHILIS

SYNONYMS: *Lues venerea*, *pox*, the French disease

Syphilis is an infectious systemic disease due to the *Treponema pallidum* (*Spirochaeta pallida*). The disease is chronic and characterized by three distinct stages, known respectively as the primary, secondary and tertiary stages of syphilis. In the absence of clinical and spinal fluid manifestations the disease is referred to as latent syphilis

with Giemsa's stain and by silver impregnation. Spirochetes are present in all of the body fluids, including the semen, in the early stages of the disease; however in the late stages of the disease they are usually absent.

Modes of Infection The disease is acquired in the majority of cases by sexual contact although few cases are



Fig 520: Left Chancre Right Giant Chancre Of penis.

Etiology Syphilis is caused by infection with the *Spirochaeta pallida*. This is a delicate, anaerobic and actively motile spiral shaped organism measuring about 8 to 10 micra in length and possessing from six to fifteen closely and regularly arranged spirals. It is readily destroyed by exposure to air and to weak antiseptics. Its presence in the tissue fluid of the chancre and secondary lesion can only be demonstrated under darkfield illumination or by staining

congenital. Abrasion of the skin and mucous membrane is the usual avenue of infection. The primary lesion is on the genital organs in 85 per cent of patients. Inoculation may result from kissing infected persons or through the use of contaminated drinking cups and table utensils, shaving and smoking accessories, pencils, telephone instruments, razor blades, surgical and dental appliances and from tattooing and vaccination. The lesions of early syphilis (chancre mu-

cous patches, and condylomata) are the most dangerous for infection in ordinary social and occupational contacts.

Primary Stage The chancre, or primary lesion, of syphilis develops at the site of inoculation within two to six weeks from the moment of infection the



Fig. 521 Multiple Chancres.

average being four weeks. The typical chancre is a well defined, moist, slightly elevated, eroded, reddish lesion with an infiltrated cartilaginous base. The surface may be covered with a grayish membrane. Usually chancres are single although there are occasionally multiple. They are usually located on the genitalia, although they may be extragenital. The sites of predilection are the glans penis, the corona, the shaft of the penis and the labia, or the external uterine os in the female. Intraurethral chancres are characterized by a urethral discharge and local induration. The most frequent extragenital locations are the lips, mouth, and nipples. The fingers of physicians and nurses are often the site

of chancre. The infection has been acquired without a primary stage from blood transfusion.

The tonsillar primary sore is accompanied by marked cervical adenitis. Chancre of the umbilicus in the newborn has also been reported. Mixed chancres result from infection with both *Spirochaeta pallida* and *H. ducreyi* bacillus. They are characterized by early and painful lymphadenitis, and a tendency to extensive ulceration.

The early diagnosis of syphilis depends on the discovery of the chancre and the *sprochaeta* in the chancre. Repeated recourse to dark field examination is most important as it makes for an unmistakable diagnosis before there are any serologic symptoms.

Diagnosis of Chancre A chancre may be confused with herpes simplex, chancroid, and carcinoma. A positive dark field is the only truly diagnostic feature of a syphilitic chancre. Puncture of a lymph node is necessary when phimosis is present, thereby preventing easy access to the chancre itself. Treatment of syphilis must be deferred until the diagnosis can no longer be doubted.

Varieties of Chancre *Frosted chancre* is a small, circular superficial smooth ulcer with a slightly indurated margin. Its color may be dark red or gray. The lesion emits a slightly purulent discharge. The *hard chancre* (hunterian chancre) is a common type characterized by a markedly indurated base with a sloping margin, giving a funnel-shaped appearance and emitting a sanguineous discharge. The *indurated papulelike chancre* is characterized by a small, flat, dark red papule with a slightly indurated base and some scaling. *Rare types* of chancre are: the herpetiform, the "silvery spot," and diffuse painless syphilitic edema occurring chiefly on a long pre-



Fig. 522: *Left:* Chancre Of left vulva. *Right:* Chancre Of left leg. Pea sized, noneroded papule. Darkfield positive on puncture and examination of serum. Three week duration. Note its insignificant character.



Fig. 523: *Genital Chancres. Left:* Of mid lower abdomen. *Right:* Of finger.

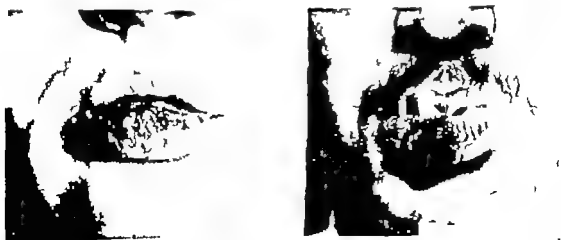


Fig. 524: *Chancres. Left:* Large, ulcerative, of lip. *Right:* Ulcerative crust-covered.

poor or vul. *Mixed chancre* is a chancre coexisting with chancreoid.

The local lymphadenopathy accompanying a chancre is characterized by



Fig. 325 Chancre Of the lower lip, with secondary eruption on face Wassermann plus 2.

painless induration and enlargement of the regional lymph nodes. The lymph nodes are sharply defined and movable.

The diagnosis of primary syphilis is established by laboratory procedures. The clinical findings and history are confirmed by demonstrating the *Treponema pallidum* in smears and by follow up serology. Several smears taken for five successive days may be necessary to establish infection. Failure to find the *Treponema pallidum* is not regarded as a final diagnosis because the organism may have completely disappeared from the suspected site of infection. This explains the necessity of serologic follow up. Weekly blood studies for at least twelve weeks in the absence of positive bacteriologic findings will provide a definite and sure diagnosis. It has been shown that Wassermann tests of the serum and exudates of chancre may show positive serologic findings in the case of a negative blood Wassermann reaction. This last test is considered a



Fig. 326 Chancre Left On mucosal side of lower lip. Right Girl aged sixteen Hypertrophic type Only slight infraauricular node enlargement.

valuable adjunct in diagnosing syphilis

Secondary Stage The secondary stage of syphilis is the period of its evolution. Local and constitutional symptoms appear about six weeks following the chancre. Cutaneous lesions may be erythematous papular follicular pustu-



Fig 527 Syphilis. Secondary stage

lar acuminate and pigmentary. Papules occurring about the perineum, vulva, anus, and scrotum are moist. These lesions are polymorphous and comprise macules, papules and pustules. They are at first bright red, finally turning to a dull red, coppery color. They may resemble the color of raw ham. The lesions have a tendency to appear in groups with a circular, semicircular or annular arrangement. Vesicular lesions, pain and itching are rare except in the Negro race. The sites of predilection are the face (forehead, areas about the nose and mouth), feet, hands (palms), anus, and genitalia. Alopecia is common

Onychia and paronychia may occur. The rupial syphilid is a pustuloulcerative lesion characterized by brownish or purplish crustaceous bulky crusts which resemble an oyster shell. The lesions are discrete and begin as papules or maculopapules. The sites of predilection are the face and extremities.

Mucous patches occur on the mucous membrane and mucocutaneous surface. The lips, buccal mucous membrane, palate, tongue, anal and vulvar mucosa are sites for mucous patches. Mucous patches are covered by a removable gray or grayish white diphtheroid membrane and when this is removed a red, raw



Fig 528: Secondary P. polar Syphilides. Of recent early syphilis.

occasionally bleeding surface is exposed. They may become ulcerated as a result of secondary infection. Mucous patches may develop into condylomata. Mucous patches in the mouth must be differentiated from aphthous stomatitis and herpes simplex. Lesions of aphthous stomatitis are sharply defined, extremely sensitive and evanescent. Herpes sim-

plex of the mucous membrane often gives a history of previous attacks and a negative result to dark field examination.

Syphilitic angina is characterized by a dark red colored throat. A definite line

of demarcation between indurated and healthy tissue is a valuable diagnostic sign of this angina.

The constitutional symptoms consist of slight fever, malaise, headache, mental depression, anorexia, enlarged spleen, bone pains, and anemia. These symptoms may be severe or mild or may be completely absent. Iritis, vertigo, tinnitus, and deafness may be present. A generalized painless lymphadenopathy is a diagnostic sign of secondary lues.

Diagnostic features of secondary syphilis are multiplicity of lesions, widespread distribution and discreteness of the lesions, polymorphism, absence of itching, except in follicular eruptions and in the Negro race, tendency to form circular, annular, oval or reniform lesions and to group in serpiginous or corymbiform arrangement, mucosal involvement, sore throat, generalized lymphadenopathy, constitutional symptoms, and positive serology.

Tertiary Stage Tertiary syphilis may appear a few months after the symptoms of the secondary stage have elapsed, or it may appear as late as fifty years after the initial primary lesion.



Fig. 529 Secondary Syphilis. Large papular syphilides.



Fig. 530 Left Periorificial Papular Secondary Syphilis. Right Periorificial Secondary Syphilis.

valuable adjunct in diagnosing syphilis.

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Fig 528: Secondary Papular Syphilides. Of acute early syphilis.

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FIG. 532 Grouped Folliculopustular Secondary Syphilis. Note characteristic grouping over upper abdomen.

Nodular or Tubercular Syphilids It is characterized by firm, copper-colored lesions the size of a split pea or larger covered by adherent scales exposing superficial ulceration on removal. Individual lesions never attain great size. The lesions tend to form rings and to undergo involution with the development of new lesions beyond them, so that characteristic serpiginous patterns are formed. Serpiginous patterns occur

frequently on extensor surfaces of the arms and on the back of the trunk. Individual serpiginous patches consist of nodules in different stages of development. It is common to observe scars and areas of pigmentation together with fresh, ulcerated lesions. This process may continue for years if untreated until large areas of the skin become involved. The depth of lesions and the extent of necrosis vary greatly. The



Fig 531: Maculopapular Squamous Secondary Syphilis.

The average length of time for tertiary syphilis ranges from three to five years after the appearance of chancre

The cutaneous lesions of tertiary syphilis may be nodular gummatous, or papulosquamous. They may be single or multiple and superficial or deep. These

lesions are characterized by indolence, painlessness, grouping in configurate arrangement and tendency to ulcerate and form scars. Two main groups of cutaneous tertiary syphilis are recognized, namely tubercular syphilide (nodular syphilide) and gumma.



Fig. 334 Secondary Palmar Syphilis. Of acute early syphilis. Note the maculopapular spots.



Fig. 336 Syphilis. Secondary stage "rupia."



Fig. 335 Syphilis. Late secondary lesion on nose.



Fig. 337 Secondary Syphilis. Note circinate lesions on face and mucous patches of lower lip.



Fig. 533: Large Papulo-squamous Secondary Syphilide

process may leave no perceptible scars or it may lead to marked destruction. Loss of pigment and subsequent hyperpigmentation may become evident. Nodular syphilide is of three to six months duration although one occasionally encounters lesions lasting over many years.

Tubercular syphilide occurs usually in the third or fourth year of the disease,

rarely before the end of the first year.

Tubercular syphilide is differentiated from lupus vulgaris which begins in childhood with the persistence of lesions throughout adult life. The patches of lupus vulgaris consist of soft nodules, the color of "apple jelly." They tend to ulcerate, possess undermined and irregular margins, have thin crusts, and leave



Fig. 512 Annularopapular Secondary Syphilis.



Fig. 514 Acute Early Syphilis. Corneal keritis. Papular syphilides on hairline.



Fig. 513 Acute Early Secondary Syphilis. Subpreputial erosive syphilides. Scum from the grayish, rounded, eroded lesions was teeming with *Treponemata*.

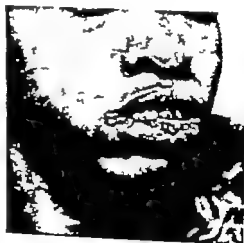


Fig. 515 Acute Syphilis. Syphilitic split papule at right angle of mouth and mucous patches on lip. Also note characteristic papule in right nasolabial groove.



Fig. 530: Syphilis. Secondary Stage
Not the additional unusual location
between the toes



Fig. 540: Sycoform P papular
Secondary Syphilis.



Fig. 539: Syphilis. Secondary stage.
Condylomata lata.



Fig. 541: Lat Secondary Syphilis.
Bilateral split papules.



Fig. 348 *Left* Annulopapular Late Secondary Syphilide *Right* Late Secondary Syphilis. Annulopapular lesions.



Fig. 349 Parulisiform Late Secondary Syphilides. Relapsing secondary lesions, about fifteen lesions in all, an asymmetrical, sparse eruption.



Fig. 350 Recurrent Late Secondary Syphilides. Rupial type and tertiary allures; lesions few in number and asymmetrical located.



Fig. 546: Late Acute Syphilis. Fram-
besiform relapsing syphilides.

phable white scars surrounded by hyperpigmentation. Active nodules do not occur in syphilis but appear within the scars of lupus vulgaris.

Gumma *Gummatosa* may appear singly or may be widely disseminated lesions, or may assume serpiginous patterns resembling lesions of nodular syphilide. A gumma may be localized in the skin or may originate in the subcutaneous tissue in which it breaks down and extends into the skin. Individual lesions begin as small nodules enlarging gradually to attain a diameter of 1 or more cm. Extensive central necrosis may lead to the formation of deep ulcers. Progress of necrosis may take place in one area of the gumma while healing proceeds in another. This leads to the formation of kidney shaped lesions which are diagnos-



Fig. 547 *Condylomata lata*. Left Secondary syphilides. Right Perioral and perianal moist papules.



Fig. 554 *Left* Ulcerated Gumma. Of upper third of leg, anteriorly. Crust has been removed to expose upper portion of the ulcer. *Right* Superficial Gummatous Ulcerations.



Fig. 555 Subcutaneous Gumma. Of lower right cheek. Site of plums and involing soft part of cheek. Of six cms diameter.



Fig. 556 Ulcerated Gumma. Of lymph node.



Fig. 551 Agminate Nodular Tertiary Syphilide



Fig. 552: Ulcerated Gumma. Of vul a.



Fig. 553 Gumma. Above philtrum. These lesions healed almost completely under nonspecific therapy; his syphilis and positive serologic evidence of syphilis, were of course not affected.



Fig. 559 Tertiary Syphilis. Showing characteristic tendency to heal in one point and to spread centrifugally



Fig. 560 Tertiary Syphilis. Psoeiform, small, nodular tertiary syphilide with syphilitic palmar hyperkeratosis. Ten years duration. Treated by all previous consultants as an eczema.



Fig. 561 Syphilis. Characteristic kidney-shaped, tertiary lesion on leg.

tic of gumma. The lower leg is the site of predilection for isolated gumma where deep punched-out ulcers arise surrounded with an extensive infiltration simulating lesions seen in varicose veins. Gumma may arise in connection with the perios-



Fig 557. Gumma.

teum and bone. A common location for periosteal and bone gumma is about a sternoclavicular joint. The calvarium is another rare but characteristic location where multiple deep ulcers appear. Healing in the bone occurs leaving cup-shaped bone defects. An unusual fibroid type of gumma known as "juxta articular nodule" consists of fibrous cutaneous and subcutaneous nodules occurring near extensor surfaces of joints, especially about the olecranon process. The composition of "juxta articular nodule" is so firm to palpation as to suggest osteoma.

Gumma is differentiated from epithelioma, varicose ulcer, erythema induratum, leprosy and tuberculous ulceration. Epithelioma (rodent ulcer) is charac-

terized by a waxy looking hard, rolled in" border and is of longer duration than gumma. Varicose ulcer is irregularly shaped and possesses an undermined border while gummatous ulceration is usually circular and has a punched-out appearance. Varicose ulcer occurs on the lower part of the leg and accompanies varicose veins, while gummatous ulceration is common over the ankle, where it is arranged in configurate patterns due to the appearance of multiple gummata. Erythema induratum has predilection for the back of the legs, where multiple indolent nodules appear and undergo central necrosis. Healing



Fig 558. Gumma. Extensive of tip of nose, upper lip, and alar nail. Return to normal was almost complete following antisyphilitic therapy.

of erythema induratum tends to form depressed scars without producing the characteristic gummy exudate of gumma. Erythema induratum occurs principally in women in their early twenties, at which age the appearance of gumma is

unusual. Ulceration in leprosy may simulate gumma but the peculiar feature of the former is the likelihood of a surrounding zone of anesthesia and the association of other manifestations of the disease. The ulceration of a broken-

Leukoplakia is observed in many luetic patients. It is not *per se* a luetic process but a coincidental implantation of syphilis upon an already present chronic irritation arising in connection with smoking chewing tobacco, or ill fitting dental restorations. Advanced leukoplakia may be associated with interstitial glossitis in tertiary lues producing firm thickening of the tongue and hypertrophy of the lingual papillae which become covered with a white verrucous covering giving rise to what is known as cobblestone tongue.

The hard palate is occasionally perforated in its center by gumma extending



Fig. 566 Syphilis. Tertiary stage.

d gumma is readily distinguished from tuberculosis by its outline, which is crescentic, kidney-shaped, or serpiginous.

Late Mucous-Membrane Lesions. The tongue is a favorite site of gumma. Gumma of the tongue attacks the central portion and rapidly breaks down to form the typical punched-out ulcer with irregular soft border. Carcinoma of the tongue is differentiated from gumma by the fact that carcinoma is invariably hard and selects the border and floor of the mouth. Gumma is free from pain while carcinoma is very painful.

Diffuse enlargement of the tongue (macroglossia) occurs in advanced stages of tertiary syphilis.



Fig. 567 Destructive Tertiary Syphilis. Center of face. Result of pharyngitis in tertiary syphilis.

ing into the velum with destruction of uvula and necrosis of the nasal bones.

Diagnosis. Diagnosis of syphilis is made by the case history, physical examination, and laboratory findings (serology and dark field examination). In this regard it is well to remember that



Fig. 562: Syphilis. Tertiary stage



Fig. 564: Tertiary Rosacea-like Syphilis. Limited to the face. Duration two years. Wassermann plus 4. The lesions are actually small gummas, some of which are still active especially on right cheek and bulbous area of nose while others had characteristically healed, leaving scars where the skin had been destroyed.



Fig. 563: Syphilis. Tertiary stage. Resultant cicatricial alopecia.



Fig. 565: Tubercular Syphilis. Of three or more years duration.

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The hard palate is occasionally perforated in its center by gumma extending



Fig. 567 Destructive Tertiary Syphilis. Center of face. Results of phagedenic tertiary syphilis.

ing into the velum with destruction of uvula and necrosis of the nasal bones.

Diagnosis. Diagnosis of syphilis is made by the characteristic physical examination and laboratory findings (serology and dark field examination). In this regard, it is well to remember that



Fig 568: Tertiary Syphilis. Neurosyphilide (Unna). Nonpurulent, non-caly, nonfiltrated, pale pink macule often annular extremely persistent, single or multiple lesions.



Fig 569: Tertiary Syphilitic Adenopathy. Three months duration. Under treatment for either tuberculous or pseudoleukemia. Wassermann and Kahn 4 plus. Rapidly disappeared under antisyphilitic therapy.



Fig 570: Syphilitic Bursopathy (Verneuil). The elevated, fungating mass is characteristic of the associated gummatous condition found in some instances in the late stages. In other instances, there are deep ulcers. Ordinarily Verneuil bursopathy is characterized by persistent, inflammatory noninflammatory tense swelling of a joint.



Fig. 571 Ulcerated Gummous Infiltration. Of upper lip, three months duration. Mistaken for cancer and given radium therapy. There is well-marked, sclerous growth just anterior to the circumoral papillae plus universal smooth atrophy of the buccal papillae. The tongue condition should have led to suspicion of syphilis. No routine examination is complete which does not include an examination of the buccal and buccal masses.

biologically false positive serologic reactions are found in malaria, acute respiratory diseases, infectious hepatitis, mononucleosis, transfusions with positive blood, and after vaccination for smallpox. They are, however apt to be weakly positive and on quantitative serologic studies at regular intervals, a spontaneous progressive decline in positivity occurs.

Prophylaxis Prophylaxis against syphilis may be local or systemic. Local prophylaxis is the most important and may be either chemical or mechanical.

The correct use of the condom before and during intercourse is the safest preventive of infection. Condoms, however do not protect extragenital areas; therefore, it is advisable to use the condom in addition to chemical prophylaxis. The Army and Navy prophylaxis is usually



Fig. 572 Heredisyphilic Gangra.



Fig 573: Syphilis. *Left* Hutlinson's teeth. *Right* Hutchinson teeth. Typical peg-shaped, notched, centrally separated, upper central incisors.



Fig. 574: Syphilis. Hutlinson's teeth



Fig 575: Congenital Syphilis. Sabin, with active tertiary lesions of skin over tibia.

cessful. The following procedure is recommended:

1. Urinate.
2. Wash the genital- scrub genital area, thighs, hands, face and all exposed part of the body thoroughly for two minutes with soap and hot water. Pay special attention to the folds of the skin and mucous membranes.



Fig. 576. Rhinogates. Note the numerous labial and perilabial, radiating scars. Note screw-driver incisions.

3. After thoroughly drying, rub a calomel ointment (33.3 per cent) into all exposed parts. Allow the ointment to remain on for five hours.

Chemical prophylactic sets are available to men of the Army and Navy and may be purchased in drug stores by them; it is available as "Sanitube" or "Andron." Chemical prophylaxis is most effective when applied within a half hour after exposure and may be of some value later, up to 1 hour after exposure.

Prophylaxis in the female consists of cleansing the external genitalia and em-

ploying a louche of bichloride of mercury 1:2000. The external parts are rubbed with 33.3 per cent calomel ointment. The injection of mapharsen or the use of any anti-syphilitic is not recommended unless the patient is willing to take anti-syphilitic treatment as outlined under penicillin therapy (p. 715).

The prevention of syphilitic infection of the offspring is accomplished by the proper treatment of the pregnant woman.

Treatment in Early Syphilis

The rationale of the treatment for syphilis is the destruction of the spirochetes without injury to the patient. Few diseases respond more readily to treatment than syphilis, and few diseases have been more often wrongly or insufficiently treated.

From the diagnostic standpoint, early syphilis is divisible into three stages: (1) seronegative primary syphilis (chancre negative blood Wassermann), (2) seropositive primary syphilis (both chancre and positive Wassermann), and (3) early secondary syphilis (the appearance of subjective and objective evidences of generalized constitutional involvement of which mucocutaneous lesions may be a part). It is definitely known that the prospect of cure drops about 20 per cent if one waits to begin treatment until the blood tests become positive or the secondary manifestations appear. It is, therefore, desirable to begin treatment in the seronegative primary stage if possible; but this does not imply the institution of treatment before it is definitely known whether a suspicious genital lesion is actually the primary sore of syphilis. It is mandatory to make early and repeated darkfield examinations of the local lesion and begin treatment as soon as one such examination is positive. Failing to obtain a positive dark field, it



Fig. 573: Syphilis. *Left:* Hutchinsonian teeth. *Right:* Hutchinsonian teeth. Typical, peg-shaped, notched centrally separated, upper central incisors.



Fig. 574: Syphilis. Hutchinsonian teeth.



Fig. 575: Congenital Syphilis. Rubber 1 in. with acetyl tertiary lesions of skin over tibia.

ven at about the middle of the forearm. Each forearm is used on alternate days. The patient is kept in bed during the period of treatment only. While undergoing the treatment, a high caloric diet is given with emphasis on an increased carbohydrate intake.

Since the introduction of this slow intravenous therapy a rapid intravenous drip method has been introduced. In this method, a small syringe is used and 240 mg. of mapharsen dissolved in 1000 cc. of 5 per cent dextrose solution is given in one to three hours.

Thomas Schoch and others give one or more injections daily using the small syringe in doses varying from 80 to 100 mg. of mapharsen.

Goldblatt has treated ambulatory patients with mapharsen for thirty days, in mg 80 mg daily. In the seronegative primary cases, the results were 100 per cent cured.

The final results of these methods of treatment cannot be evaluated for some years. It is also recognized that this method of treatment is valueless in the treatment of late syphilis.

Penicillin Therapy Penicillin is the most effective and the least toxic drug so far discovered for the treatment of syphilis. Furthermore penicillin, by itself and especially if reinforced with some heavy metal therapy, gives great promise of curing patients with acute syphilis, even when complicated by a symptomatic or early meningeal neurosyphilis. In any event, penicillin renders the patient rapidly noninfectious and causes rapid resolution of lesions. The therapeutic effectiveness of penicillin appears to be greatly influenced by the time-dose relationship of its administration. Its activity appears to depend upon the length of time therapeutic effective levels are

maintained in the infected person's blood. The drug is actually spirochetocidal in extremely low concentrations. Such low concentrations acting over long periods of time appear to be more efficacious than high ones over a shorter period. The duration of penicillin action has been extended by suspending the drug in beeswax and peanut oil.

The Penicillin Committee of the National Naval Medical Center reports that out of 250 patients with early syphilis, 25% remained symptom free. Sternberg and Lerner analyzed the records of 1400 men with early stages of syphilis treated with a total dosage of 2,400,000 units of penicillin. There was an average satisfactory progress rate in 90.6 per cent with the best results in seronegation-chancere stages (94.5 per cent). The overall results of penicillin therapy alone, however, especially of early syphilis, are not clear at present because during 1944 and 1945 (inclusive) commercial penicillin contained much penicillin K, which is rapidly destroyed in the body and is relatively noneffective against *Spirochaeta pallida*. Commercial penicillin of today, however, has a high percentage of penicillin G which is slowly destroyed in the body and destructive to *Spirochaeta pallida*. Since commercial penicillin consists of varying mixtures of five known fractions and since exact methods to determine the amount of each fraction in a given batch of commercial penicillin is not available, the National Research Council advises large doses in the treatment of syphilis. The end results of the therapy will require years for its evaluation. Nevertheless, at present, the anticipated cure rate in acute syphilis with the dosage advised is 90 per cent or better.

becomes necessary to do Wassermann tests on the fluids from the lesion or adjacent lymph nodes

The cooperative patient will comply with the following requirements (1) Abstain not only from sexual intercourse but from all physical contact with other individuals or any articles used by others, such as pipes, towels, shaving equipment etc (2) scald dishes after use (3) burn all dressings (4) take good care of the teeth and when visiting the dentist tell him of the infection (5) abstain from smoking and drinking (6) report pimples, hemorrhoids, cough, colds, and all other illnesses and indispositions to the physician

If the patient is not treated in the beginning of the infection serious late syphilis may be expected in about 25 per cent of cases, benign late syphilis in 15 per cent, latent syphilis in 30 per cent, and spontaneous "cure" in 30 per cent. If the patient is given early treatment continuously and adequately, complete cure may be expected in about 90 per cent of cases, and symptomatic cure but with retention of a positive serologic reaction in another 5 per cent of the patients

Heavy Metal Therapy The principles of treatment in early syphilis are predicated on early diagnosis, as well as on the use of properly chosen drugs and continuous treatment without a rest period of from twelve to eighteen months and with repeated serologic examination during the life of the patient

Earlier methods of treatment have been replaced by continuous treatment, namely, alternating courses of an arsenical and a heavy metal in proper dosage and over long periods of time without interruption. The following outline of treatment is recommended for early syphilis.

Twelve weekly injections of an arsenical and bismuth subsalicylate

Six weekly injections of bismuth subsalicylate

Ten weekly injections of an arsenical

Eight weekly injections of bismuth subsalicylate

Ten weekly injections of an arsenical

Ten weekly injections of bismuth subsalicylate

Eight weekly injections of an arsenical

Twelve weekly injections of bismuth subsalicylate

During the initial period it is considered good practice to give both arsenic and bismuth concurrently. The injections are given at intervals of several days, however bismuth and arsenic should not be given on the same day.

Arsphenamine is better than other arsenicals, however mapharsen and neoarsphenamine are more quickly prepared and administered and are therefore preferred. The dose of mapharsen is 0.04 to 0.06 gm., of neoarsphenamine 0.175 to 0.3 gm. of arsphenamine 0.4 gm. In women a smaller dose is desirable.

Bismuth subsalicylate in oil is the preparation of choice and is given in doses of 0.2 gm. of metallic salt. Iodides are given during the heavy metal course in daily doses of 2 to 4 gm. of a saturated solution of potassium iodide.

Massive doses of arsenotherapy as practiced in the Mount Sinai Hospital, New York, consist of the administration of a large dose of mapharsen during a five-day period. The technique of the "five-day drip" consists of the administration of 240 mg. of mapharsen dissolved in 2400 cc. of 5 per cent dextrose solution delivered from a properly sterilized gravity apparatus at the rate of 3 cc. per minute on each of five consecutive days. To the dextrose solution 200 mg. of ascorbic acid is added as a capillary wall protectant. The dose is delivered in eleven hours. A standard 20-gauge 1½-inch needle is inserted full length in the

nerve deafness.) In these, a full course though a shorter course may be ample of fever therapy, preferably malaria should also be given concomitantly.

In syphilitic aortic disease aortic regurgitation, aneurysm in particular and in hepatic syphilis, large initial doses are preferably avoided. Penicillin in the chronic complications of syphilis is an adjunct to the older methods, but its exact place in the treatment of these complications is still to be decided.

Penicillin has been highly efficacious in the syphilitic pregnant patient, for the fetus in utero and especially in those women with recently acquired syphilis.

Administration of Neosarsphenamine, Mapharsen, and Bismuth Subsalicylate. Before starting the arsenical course, careful physical and urine analyses should be made, and the patient's complete history obtained. Evidence or history of skin hypersensitivity such as chronic urticaria, eczema, seborrheic dermatitis, or frequent "rashes," will indicate the necessity of starting with much lower than usual doses. There is also evidence that untreated syphilis adversely affect the course of pulmonary tuberculosis. A smaller dose of arsenic is advisable and if the tuberculous state is far advanced, treatment should be started with bismuth. If the patient is pregnant or has had a liver or cardiovascular involvement, special

care must be employed in the choice of drug and their dosage. In hyperthyroidism, for example, such explosive reactivity to the arsenphenamines that be careful to control the hyperthyroidism before beginning antisyphilitic therapy. History of marked easy "bruising" should make one very cautious in the use of arsenphenamines, and evidences of purpura on mucous membranes or skin should contraindicate the use of these

drugs. Presence of nephritis and diabetes demands caution and the use of smaller initial doses. Primary anaemia necessitates dosage reduction in the beginning, but secondary anaemia is usually quickly benefited by the arsenicals. Arthritics and hypertensive cases are often helped by antisyphilitic treatment. Optic or acoustic nerve involvement should be treated for three to six weeks with bismuth.

Diet During Treatment. The meal preceding the injection and the next one following it should consist of toast and coffee or tea, and nothing at all should be placed in the stomach for at least two hours before the injection. Until recently it had been the almost universal custom to place the patient on a high carbohydrate and low fat and protein diet. This is accomplished by increasing the intake of bread, potatoes, and cereals and limiting the intake of meat, fish, eggs, milk and butter. Lately, however, this regimen has been reversed and more protein and fat is given, while the carbohydrates are diminished. The experimental study of Craven in 1931 which first suggested the trial of this new type of diet, has been confirmed by Messinger and Hawkins, who found protein more protective than carbohydrate and the inclusion of fat distinctly deleterious. Individuals who suffer from diarrhea while on arsenical treatment are much helped by reducing or eliminating the consumption of fruits and coarse vegetables.

Preparation of Neosarsphenamine Solution. The powder should be less than canary yellow; if the color approaches red and the drug is lumpy or solidified, it should not be used. Pour about 5 cc. of sterile, distilled water into a sterilized beaker, dip the ampule into alcohol to detect a possible crack, file it open and

Acute Syphilis This term generally refers to the first five years of the infection and includes the following:

- 1 Seronegative-chancere stage
- 2 Seropositive-chancere stage
- 3 Early secondary stage
- 4 Late or relapsing secondary stage
- 5 Pretertiary stage—symptomless except for serologic evidences

Although an ideal treatment schedule for acute syphilis has not yet been evolved the dose advised at present is 600 000 units dissolved in aqueous or isotonic saline solution given intramuscularly every three hours day and night for a total of 12 000 000 units, or 300 000 units of calcium penicillin in beeswax and peanut oil morning and evening intramuscularly for ten days a total of 6 000 000 units. At the present writing it is believed that penicillin therapy should be reinforced in all stages except the seronegative-chancere phase with at least one subsequent course of twenty injections of bismuth salicylate (100 mg at weekly intervals) semimasine mapharsen therapy using one half the usual dosage (120 mg daily for five days) or a course of neoarsphenamine consisting of weekly injections to a total dosage of 12 gm in divided dose 0.45 gm to 0.75 gm. Serologic or clinical relapse requires a repetition of the therapy with penicillin and the heavy metals. The addition of a course of heavy metal therapy is based upon the fact that it is preferable to overtreat than undertreat syphilis. Likewise on penicillin alone "relapse" rates both cutaneous and serologic have ranged from 10 to 20 per cent. Finally clinical and laboratory results, when penicillin and neoarsphenamine are used concurrently have proved superior to those with penicillin alone (Eagle et al. Kolmer et al.) Severe Herxheimer reactions may occur when penicillin is given other

wise the therapy is practically complication free. In favorable cases of penicillin treated syphilis, the objective signs of the disease disappear within two to four weeks, and the blood serum evidences, as based upon monthly quantitative titration disappear in about two and one half to six months. In the seronegative-chancere patients, the blood in favorable cases never becomes positive. The results therefore are much more effective if therapy is instituted early in the course of the disease.

In all cases, monthly inspection and quantitative serologic tests are required for twelve months after therapy.

Chronic Syphilis Penicillin favorably influences the course of chronic syphilis and its complications. A total dosage of 8 to 10 million units appears to obtain best results. Penicillin is the best remedy for late oral, cutaneous, bone, and most forms of visceral syphilis.

The drug is definitely a roborant in all patients with chronic syphilis and therefore is indicated in chronic latent syphilis, not only for this reason but because of its known value in acute syphilis. However it rarely reverses the serologic evidence of the disease. Although it can do no harm in "Wassermann-fast" patients who have had prolonged chemotherapy it appears, except for its roborant effects, to be of little value in these.

Penicillin is of significant value in all forms of symptomatic and asymptomatic neurosyphilis. It favorably influences the evidences of syphilis in the spinal fluid especially the cells and globulin content many of the subjective disturbances, and even the Wassermann reaction.

Penicillin ought not to be used to the exclusion of other recognized methods, in neurosyphilis, certainly not in the parenchymatous forms (paralysis, tabes dorsalis, primary optic atrophy and late

nerve deafness) In these, a full course though a shorter course may be ample of fever therapy preferably malaria, should also be given concomitantly.

In syphilitic aortic disease aortic regurgitation aneurysm in particular and in hepatic syphilis, large initial doses are preferably avoided. Penicillin in the chronic complications of syphilis is an adjunct to the older methods, but its exact place in the treatment of these complications is still to be decided.

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Administration of Neosyphenamine, Mapharsen, and Bismuth Subsalicylate Before starting the arsenical course, careful physical and urine analyses should be made and the patient's complete history obtained. Evidence or history of skin hypersensitivity such as chronic urticaria, eczema, seborrheic dermatitis or frequent "rahes," will indicate the necessity of starting with much lower than usual doses. There is also evidence that untreated syphilis adversely affects the course of pulmonary tuberculosis. A smaller dose of arsenic is suitable and if the tuberculous state is far advanced treatment should be started with bismuth. If the patient is pregnant or is paupered, or has a liver or cardiovascular involvement, special care must be employed in the choice of drugs and their dosage. In hyperthyroidism, the doctor finds such explosive reaction to the arsenphenamines that he prefers to control the hyperthyroidism before beginning antisyphilitic therapy. History of marked easy bruising should make one very cautious in the use of arsenphenamines, and evidences of purpura in mucous membranes or skin should contraindicate the use of these

drugs. Presence of nephritis and diabetes counsels caution and the use of smaller initial doses. Primary anemia necessitates dosage reduction in the beginning, but secondary anemia is usually quickly benefited by the arsenicals. Arthritis and hypertensive cases are often helped by antisyphilitic treatment. Optic or acoustic nerve involvement should be treated for three to six weeks with bismuth.

Diet During Treatment The meal preceding the injection and the next one following it should consist of toast and coffee or tea, and nothing at all should be placed in the stomach for at least two hours before the injection. Until recently it had been the almost universal custom to place the patient on a high carbohydrate and low fat and protein diet. This is accomplished by increasing the intake of bread, potatoes, and cereals and limiting the intake of meat, fish, eggs, milk, and butter. Lately however this regimen has been reversed and more protein and fat is given, while the carbohydrates are diminished. The experimental study of Craven in 1931 which first suggested the trial of this new type of diet, has been confirmed by Messenger and Hawkins, who found protein more protective than carbohydrate and the inclusion of fat distinctly deleterious. Individuals who suffer from diarrhea while on arsenical treatment are much helped by reducing or eliminating the consumption of fruits and coarse vegetables.

Preparation of Neosyphenamine Solution. The powder should be lemon to canary yellow. If the color approaches red and the drug is lumpy or solidified, it should not be used. Pour about 5 cc of sterile, distilled water into a sterilized beaker dip the ampule into alcohol to detect a possible crack, file it open and

pour the contents on to the surface of the water allow solution to take place without shaking the beaker because agitation will increase the oxidation of the drug and thus greatly increase its toxicity. Solution made directly in the ampule is very likely to be more toxic than that made as above described. It should not require more than five minutes to dissolve the drug completely and the solution should be rejected if more than ten minutes is necessary to accomplish this. Inject as soon as possible in no case employ a solution which has been standing more than twenty minutes. Inject very slowly. The drug should be stored in the ampules in a refrigerator and the solution made at room temperature the toxicity increases rapidly above 41°F . Do not use ampules that are more than six months old.

Preparation of Mapharsen Solution Mapharsen is a white powder; it should not be used if it is gray, brown or black. Ampules should be stored in a refrigerator. The precautions against oxidation of the drug so important with neoarsphenamine are not necessary here in deed mapharsen becomes less toxic on exposure to air and therefore agitation of the solution during preparation is desirable. The solution is made by dissolving 40 mg. of the drug in 1 cc. of sterile distilled water.

Intravenous Administration of Neoarsphenamine and Mapharsen The essential steps are the following: (1) Have patient reclining on back with arm bared and extended at right angles. (2) Apply tourniquet above elbow and wipe the injection site with 70 per cent alcohol on cotton. (3) Eject all air from syringe and needle, and introduce needle into vein. (4) Draw back on plunger and be sure that a full sized stream of blood enters syringe quickly and freely. (5)

Remove tourniquet and begin the injection. (6) Inject neoarsphenamine very slowly the United States Public Health Service standard rate is 0.1 gm. per thirty seconds, but ample experience of many men has shown that much slower injection than this is advantageous; the slower the better. The use of a small-bore needle prevents rapid administration. (7) Mapharsen can be injected intravenously as rapidly as one wishes; indeed from the standpoint of preventing pain from venous spasm the faster the better. (8) At the conclusion of the injection bring the needle out quickly and apply at once a small bit of sterile cotton to the site of entry.

Intramuscular Administration of Bismuth Subsalicylate in Oil Insert the needle point down in the inner part of the upper outer quadrant of the buttock. After carefully aspirating to be sure that the point is not in a blood vessel, inject slowly and finish by detaching syringe drawing up 1 to 2 cc. of air and injecting this to prevent drawing some of the drug toward the surface as the needle is withdrawn.

Massaging deeply for several minutes, protecting the injection site by a pledget of alcohol soaked cotton aids in the distribution of the drug and lessens the incidence of severely painful reactions.

Arsenical Reactions in Early Syphilis. Nitritoid Reaction This type of reaction (not seen with mapharsen) usually begins shortly after the neoarsphenamine injection has been started or the bismuth has been placed in the buttock. There may be sudden intense pain in the back though this is very rare; however there is a quick general or blotchy flushing of the face and neck, injection of the eyes, dyspnea, cough, nausea, anxiety and occasionally vomiting. The pulse often becomes momentarily bound.

ing and then very weak and edema of the face and neck may supervene. Sometimes there is loss of consciousness with suspended respiration for a few seconds. These symptoms usually persist for half an hour or longer. The nitritoid reaction is rarely fatal.

The treatment for the nitritoid reaction is the immediate subcutaneous or intramuscular injection of 0.5 to 1 cc of epinephrine hydrochloride (adrenalin) 1:1000 solution. It is sometimes possible to prevent the recurrence of this reaction by the subcutaneous injection of 0.0008 to 0.0012 gm. ($\frac{1}{4}$ to $\frac{1}{2}$ grain) of atropine sulfate fifteen minutes before the injection, or epinephrine may be given before the injection instead of after the symptoms have appeared. Ephedrine sulfate is sometimes used, several doses of 0.025 gm. ($\frac{1}{4}$ grain) by mouth on the day of the injection is a prophylactic measure.

Lacrimation-Salvation Reaction. A reaction apparently peculiar to mapharsen consists in mild lacrimation with mild to profuse salivation beginning fifteen minutes to three hours after injection and lasting several hours. This reaction is not a serious one.

Gastrointestinal Reactions. Many individuals are much disturbed by an etherlike odor and disgusting taste while neoarsphenamine is being injected. This may be counteracted by causing the patient to smell bayrum or a perfume during the operation (the cheaper perfume is preferable because of their greater pungency). The rapid mastication of wintergreen chewing gum is equally serviceable.

The occurrence of stomatitis during arsenical administration is characterized by a reddened mucosa and chapping of the lips. Its chief significance is that it may be a warning of an existing idio-

synchrony for the drug. Gastrointestinal reactions (diarrhea) occasionally follow the use of the arsenicals; however fewer gastrointestinal reactions occur after mapharsen than after neoarsphenamine. Levin and Keddie in 1942 said that about 90 per cent of patients who had severe gastrointestinal reactions to the arsphenamines tolerated mapharsen in therapeutic doses.

Skin Reactions. Urticaria or a scarlatiniform erythema often occurs during the nitritoid reaction and subsides with it. These fleeting skin involvements do not usually require any treatment. Generalized itching unaccompanied by dermatitis and following upon the injection of an arsenical is generally looked upon as a warning signal and further arsenical treatment should not be given. "Ninth-day fever" is characterized by a scarlatiniform or morbilliform eruption occurring on the eighth to twelfth days after one of the early injections. It is associated with chills, fever, pharyngitis, vomiting, pains throughout the body, superficial adenopathy and occasionally photophobia. There may also be subsequent desquamation as in scarlet fever. The reaction is nearly always over in five to seven days, but Mour (1937) reported a case which was followed by severe neuritis and later by severe vesicobullous dermatitis and death from toxic degeneration of internal organs. In Robinson's (1938) case, which developed after the third injection of neoarsphenamine, treatment was stopped for two weeks; upon its resumption hepatitis and jaundice developed very promptly.

A "fixed" type of eruption is occasionally seen, this does not contraindicate continuance of treatment. It is characterized by the appearance of erythematous, edematous plaques at the site of injection.

The most important skin manifestation of arsenical intolerance is dermatitis exfoliativa. This reaction may occur at any time during treatment. It is characterized by the following symptoms:

(1) general flush with dry or moist dermatitis of flexor surfaces and lower parts of the body, accompanied by very severe itching; (2) usually edema and a septic temperature; (3) the blood picture shows early leukopenia, decrease in polymorphonuclear neutrophils and an increase of eosinophils up to 60 per cent (decrease in the number of eosinophils is one of the first signs of recovery); (4) the symptoms of dermatitis exfoliativa may appear a few hours after an injection and death may occur very early with intercurrent respiratory or myocardial symptoms or it may persist for many months and terminate fatally from pneumonia or nephritis.

TREATMENT. All arsenical treatment should be at once stopped. Start BAL or dimercaprol therapy (p. 721). Local treatment: purely palliative and consist of daily colloid baths and the application of a mild ointment such as aquaphor. If infection is present a 5 per cent sulfathiazole ointment is recommended. Severe cases need hospitalization and intravenous therapy of calcium sodium hypophosphite and ascorbic acid (vitamin C). Edward D. Jenkins, Past Assistant Surgeon of the U. S. Public Health Service, believes that histamine therapy hastens recovery in postarsenical dermatitis. The use of routine measures plus histamine therapy will give better results than histamine alone.

The prognosis of arsenical dermatitis is guardedly favorable.

In a small proportion of individuals with untreated secondary syphilis, jaundice appears merely as one of the evidences of the generalization of the early

toxemia, however in individuals undergoing treatment with one of the arsenicals, this symptom occurs with much greater frequency.

Occasionally acute yellow atrophy of the liver develops and is characterized by chills and fever, abdominal pain and liver tenderness, vomiting, delirium, prostration, coma and death.

Early syphilitic hepatitis with jaundice in an untreated patient should be treated with bismuth for four to six weeks followed by small doses of arsenic, although a few recent reports of isolated cases treated with arsenicals from the beginning showed no apparent harm.

Hematopoietic Reactions. Arsenic injuries to the blood-forming apparatus are rare. If these reactions do occur they manifest themselves either as thrombocytopenia with typical purpuric or external hemorrhagic features, granulocytopenia, presenting the picture of a fulminating agranulocytosis or aplastic anemia with the characteristics manifested when all the cellular elements of the blood have been affected. Symptoms may appear after any injection in any one of the courses. Frequent blood examinations in every patient under treatment with any of the arsenicals, even in the absence of untoward manifestations are recommended and the immediate discontinuance of the drug as soon as such symptoms as malaise, pallor, itching of the skin or purpuric signs are noted.

Minpharsen usually will not cause a purpuric reaction and this drug may safely be substituted for neoarsphenamine in individuals who have experienced an attack of agranulocytosis.

Nervous System Reactions. Hemorrhagic encephalitis may occur without any warning signs after any injection although it more often follows the earlier

injections. It is very rare and often fatal. The symptoms usually develop two or three days after treatment and consist of headache, giddiness, apprehension, delirium, vomiting, urinary and fecal incontinence, convulsions, coma, and death in forty-eight hours.

Treatment. This consists of the use of cyanoephthine hydrochloride (adrenalin) 2 cc of 1:1000 subcutaneously every two hours until improvement is noted sodium thiosulfate subcutaneous injections, 500 cc of 25 per cent dextrose solution intravenously twice daily, magnesium sulfate 1 gm. (15 grains) and morphine sulfate, 0.015 gm. ($\frac{1}{4}$ grain) as anticonvulsant and sedative. 1 mlm procedure is reported to if there is a bulging of the optic disks.

Meningitis and encephalomyelitis are even more rare and more often fatal than encephalitis. A mild degree of meningeal irritation, usually going no further than the development of numbness and tingling in the extremities, is not rare during administration of arsenicals, neoarsphenamine being a frequent offender in this respect. Severe cases of true multiple neuritis are extremely rare; sodium thiosulfate is often given in these cases and arsenicals are omitted from subsequent therapy.

Hershheimer Reaction (The repetitive shock). This is characterized by erythema of the syphilitic lesions and occurs on early syphilis anywhere from a few hours to a day after the first injection of an arsenical. It occurs more abruptly than mapharsen than with neoarsphenamine or bi-muth. The reaction disappears slowly during several succeeding days and very rarely recurs following subsequent doses. Hershheimer is small of build and is a very early syphilis because of the nature and location of the lesions but the liability of its occurrence

constitutes one of the reasons for making the first dose of an arsenical smaller than the subsequent doses in late syphilis with its numerous visceral lesions. Very grave symptoms may accompany such a reaction, which fact often dictates therapeutic approach to late syphilis through preliminary use of heavy metals.

Preventive Precautions. If treatment is continued and no precautionary steps are taken at all, 60 per cent of individuals will react again. If it is necessary to continue arsenical treatment, changing the drug is the most successful method of avoiding further trouble. Next best to changing the drug is the reduction of subsequent doses. The best assurance of avoiding a further reaction is to omit the next dose.

The development of dimercapsol or B.A.L. (British anti-lewisite) has made available a new effective antidote to arsenic and mercury. It is of value in overdosage arsenical fever, arsenical dermatitis, hemorrhagic encephalitis, blood dyscrasias, and toxic hepatitis. The best results are obtained when the drug is given early. Eagle advises 2.5 mg per kilogram of body weight (for mild cases) 5 mg per kilogram (for severe cases). Injections are given every four hours for six doses, then one injection every six hours for four doses, and finally one every twelve hours for ten days or until recovery. The drug is supplied in 10 per cent solution, and on a 5-mg per kilogram percentage, a person weighing 60 kg would receive 1.8 cc per dose.

Side effects may occur within a half hour but are temporary on the doses advised. They are combated with sedatives.

Accidental Paravenous Infiltration of Neoarsphenamine or Mapharsen. Treated by withdrawal of needle and follow by the immediate injection of 20 to

30 cc. of physiologic saline solution containing 0.5 per cent procaine hydrochloride into and around the infiltrated area. Then complete the arsenical injection at another site. Cold compresses are usually helpful in the beginning; later hot compresses of saturated solution of magnesium sulfate may be used.

Preparations for Oral Administration. Capsules of bismuthinol mass (soluble sodium bismuthate) have been used in the treatment of syphilis. Each capsule contains the equivalent of 150 mg. of bismuth. The drug is well tolerated and if given in therapeutic effective doses it causes rapid involution of early syphilis lesions and disappearance of the spirochete in about four days. Since clinical and serologic relapses have not been prevented it is not recommended as a routine treatment.

Bismuth Suspensions in Oil. The two outstanding advantages of these preparations are that their injection causes much less pain than the injection of aqueous solutions, and the injections need be made only once a week. The following are disadvantages of bismuth injection: (1) sterile abscesses of rather large proportions may occur even a long time after the injections have been stopped; (2) embolism and infarction following injection into an artery—if the latter occurs, great pain and nearly always ultimately gangrene and sloughing out of the affected area occurs; (3) occasionally during a period of very slow absorption a pocket of bismuth may suddenly enter the circulation and produce a severe stomatitis.

Bismuth is very likely to cause severe reactions and even death when given intravenously. It is only given intramuscularly.

Water Soluble Salts for Injection. These preparations are more rapidly and

more regularly absorbed than the oily suspensions, which is both an advantage and a disadvantage—advantageous in that it enables quick results to be attained; disadvantageous in that injections must be administered three in the week and are difficult to use even in office practice except in unusual instances. Furthermore these aqueous solutions are very apt to cause trouble.



Fig. 577: Cutaneous manifestations of accidental, local embolism following intragluteal bismuth injection. (Courtesy of D. Carroll S. Wright.)

some stomatitis if they are pushed too rapidly. An advantage, on the other hand, is that there is no danger of embolism or infarction following these injections which, however, are much more painful than are those of the oily suspensions.

Local reactions consisting of pain and tenderness are more often experienced with the soluble bismuth tartrate and

less frequently following the use of oil soluble bismuth compounds

The symptoms of intra-arterial injections of bismuth (local arterial embolism) consist of severe local pain, localized ischemia and purpura. The site of injection is edematous, a bluish, network-like appearance of the skin develops, and the area often becomes necrotic. In some, pain develops several hours after the injection. Treatment consists of application of cold compresses the first few hours followed by hot compresses. It may become necessary to surgically remove the affected area. Local arterial embolism is not apt to occur if after the needle is in place a paration indicates the tip is not in a vessel. Accidental intravenous injection is a more serious complication. Pulmonary embolism and bismuth poisoning are the usual after-effects of this accident. Pulmonary embolism results from the intravenous injection of bismuth in oil. The symptoms of embolism are a feeling of weight on the chest, uncontrollable coughing and occasionally shock which may be fatal. The symptoms of bismuth intoxication are extensive purpura, stomatitis, nephritis, and occasionally a terminal bronchopneumonia. The majority of patients receiving bismuth develop a bluish-gray pigmented film on the gums. It rarely is accompanied by stomatitis and is not a contraindication for treatment. Bismuth gripper is a rare reaction of bismuth therapy, characterized by malaise, slight fever, headache, and pain in the bones and joints. It often follows each injection and, if this condition continues, bismuth therapy should be stopped and mercury substituted.

Oil soluble and insoluble bismuth preparations sterilize surface tissue lesions more slowly than the soluble bismuth preparation. The latter require about



Fig. 578 Severe injury following misplaced, "intramuscular" injection. Return to normal in six months with physiotherapy. Bismuth injections were best made in the outer upper quadrant of the gluteal area with the needle inserted obliquely downward and with the beech turned outward. (Courtesy of Dr. Jacques C. Guequerra.)

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Syphilis Reactions to Mercury (1)

Direxus, occurring somewhat less frequently than with bismuth (2) renal disturbance of nephrotic nature, more frequent and severe than with bismuth (3) gastrointestinal disturbances even from 1:1000 paration not administered by mouth anorexia polyneuritis, depression even an occasional psychosis, salivary stomatitis, colic and intractable diarrhea anemia, and wasting (4) rheumatism, more severe than the grippe of bismuth (5) cutaneous reaction other than the local disturbances associated with injection, are rare.

Salivary stomatitis, gingivitis, and stomatitis are the most common local complications of mercurial therapy. Treatment of mercurial stomatitis consists of the exclusion of acid and sour foods and the increase of carbohydrates in the diet. The mouth should be rinsed after each meal with liquor alkalmi antisepticus containing 0.5 per cent zinc chloride. A triscent (tincture of kino, 30 cc. [1 ounce], and tincture of myrrh, 60 cc. [2 ounces]) should be painted on the gums or a few drops of the mixture added to an ounce of water may be used as a mouthwash, preferably after meals and at bedtime. Sodium perborate, hydrogen peroxide and sodium bicarbonate are equally useful.

Iodides. Iodides have no spirocheticidal effect. Their only indication is in the treatment for resorption of granulomatous tissue. Neurosyphilis and gummatous lesions which are relatively affected following the use of iodine. The iodides should not be given during a course of neosphenamine, however, in late syphilis and early neurosyphilis they should be given during a rest period, or while the patient is being treated with bismuth.

The contraindications to iodide therapy

are thyroid adenoma and pulmonary tuberculosis.

Dosage. Iodides are usually given orally in the form of potassium iodide.

Kali Iodid. 60.0

Aq. distillat. 60.0

Sol. Tent. fifty drops in full glass of water one half hour before meals.

If sodium or potassium iodide is not well tolerated the following organic compounds of iodine are recommended: lipiodine, momine or sajodin.

Intravenous injections of a 10 per cent sterile solution of sodium iodide is the treatment of choice in neurosyphilis. The first dose is 25 cc. daily and this is increased 25 cc. daily until 100 cc. is reached and this latter dose is continued for five weeks. The intravenous injection of sodium iodide should be preceded by the oral administration of potassium iodide for several days in order to discover any possible intolerance to the drug.

Total Amounts of Arsenic and Bismuth. The Cooperative Clinical Group studies show that the seronegative primary patient who may expect to complete his cure in twelve months, should receive a maximum of thirty doses of arsenic and the same amount of heavy metal. This means the giving of thirty intravenous injections of neosphenamine or mapharsen and about the same number of intramuscular injections of an oily suspension of bismuth subsalicylate or if bismarsen is being used, sixty intramuscular injections of that drug. The seropositive primary or early secondary patient, who must be treated for eighteen months, will have to continue the treatment until he has received half as much again of both arsenic and bismuth.

Treatment should be begun with an arsenical. If neosphenamine is being

one week while the former require several weeks. The rate of absorption and excretion depends on the chemical composition of the bismuth preparation and the individual differences in persons. Excretion of bismuth takes place via kidneys and bowels. Bismuth subsalicylate is eliminated more slowly than any of the other bismuth compounds; only 4 per cent of the amount injected being excreted within three weeks. On account of this slow elimination injections need not be given so frequently. Bismuth cannot replace the arsenamines in syphilis therapeutics; however in the intervals between arsenamine courses, bismuth enhances the natural defenses of the body and at the same time is spirocheticidal. Bismuth can also be used in combination with mercury if there is an intolerance to the arsenamines. There are few contraindications to the use of bismuth and such reactions are rarely sufficiently severe to warrant its discontinuance; however hemorrhagic diathesis of any variety, paroxysmal hemoglobinuria, cardiac decompensation and hepatic stasis, are contraindications for bismuth therapy.

Cutaneous reactions following bismuth therapy are rare. Any of the eruptions seen in postarsenical or mercury treatment may occur. Exfoliative dermatitis, erythrodermia, pityriasis rosea and urticaria are occasionally observed. The cutaneous manifestation slowly disappears and after their disappearance do not recur even though bismuth therapy is resumed.

Jaundice has been reported following the use of bismuth; however this is probably only incidental and has nothing to do with bismuth therapy.

Bismuth has a diuretic action on the kidney and albuminuria is occasionally present.

Death following bismuth therapy is rare and if it does occur it follows accidental intravenous injection.

Mercury by Inunction. The preparation to use is the U.S.P. "stronger mercurial ointment" which contains 50 per cent of mercury in a suitable base. A dose of about 4 gm. (1 drachm) is rubbed for thirty minutes into a different hairless portion of the body on each of six consecutive days, the site of inunction being previously washed with soap and water and then alcohol. A full bath with soap and water but no rub, comprises treatment on the seventh day; on the next day the second course of rubs begins. This is a very effective way of mercurializing a patient but it is difficult to get him to persist in the timed rubs, and the procedure is a very dirty one and not easy to conceal from family and associates. A very simple method of cleansing the skin after an inunction is to wipe off the excess ointment with benzine or naphtha or very quickly with a good grade of leadless gasoline. In their earlier reports Cole and Sollmann stated that this cleansing does not lessen the effectiveness of the treatment. The United States Public Health Service has employed a belt or girdle on which the ointment is smeared. Dermatitis of various sorts may arise and cause temporary cessation of treatment.

Mercury is known to be germicidal and probably has an inhibiting effect on the growth of spirochetes. Forty-five per cent of mercury is excreted via the intestines and 55 per cent via the kidneys. This fact accounts for the frequency of renal and intestinal complications following its use. Mercury by inunction is the safest effective method of administration and is the method of treatment advised when the patient has displayed an intolerance to arsenic.

uations during the first ten years following infection. Certainly two years should have elapsed from the time of the infection even though the patient has had proper medication. After the lapse of five years only 30 per cent of the patients become infected and after the tenth year none.

Treatment Resistant Syphilis The treatment-resistant syphilitic is one in whom either spirochetes, lesions, or serologic manifestations, or all three are not affected by correct antisyphilitic therapy. The arsenicals are the drugs against which resistance is most often manifested, and most of the patients are early syphilitics, though resistance may be encountered in any stage of the disease. Many authorities feel that most of these resistant cases are seen in patients who have been treated with some form of "combined" therapy. There are, however, certain complications of syphilis which do not respond to any treatment. Deafness due to involvement of the auditory nerve, primary optic atrophy, perforating ulcers, and Charcot joints do not respond to antisyphilitic therapy. The percentage incidence of seroresistance is high in early involvement of the central nervous system, but in late syphilis seroresistance is a part of the disease and may be of favorable prognostic import.

Patients with a fixed positive Wassermann reaction should be examined for cardiovascular disease, especially aortitis, and for cerebrospinal syphilis, which should include an examination of the spinal fluid.

In early Wassermann-fast syphilis, the spinal fluid should always be examined and, if found positive, the patient should be treated for neurosyphilis, and, if found negative, treatment with arsenphenamine should be given continuously for two

years. In late Wassermann-fast syphilis, the spinal fluid should also be examined and, if positive, the patient should be treated as if it were a case of neurosyphilis—if the spinal fluid is negative treatment should be continued for two years with full doses given continuously after which period periodic clinical and serological studies are made for the rest of the patient's life.

Treatment in Latent and Wassermann Fast Syphilis By the term latent syphilis, we mean that stage of syphilis where no clinical manifestations of the disease are present. Latency usually follows the secondary stage, but it may occur at any stage. Latency usually follows the secondary stage but it may occur at any stage. Latency is of undetermined duration. One out of every three patients treated for syphilis is in the latent group. This group of cases consists of those patients whose only evidence of the disease is a positive Wassermann or Kahn reaction. One must be sure that the reaction is not falsely positive. There are a large number of diseases which have a positive serology for instance, yaws has a positive Wassermann reaction in 100 per cent of cases; leprosy 40 to 60 per cent; malaria 100 per cent at some time during the infection, and infectious mononucleosis, 20 per cent.

Every effort must be made to determine whether neurosyphilis, cardiovascular syphilis, hepatic, gastric, osseous and other miscellaneous manifestations of late syphilis are present. If any of these manifestations of syphilis are discovered, the treatment is obvious; if none of them are discovered, the question remains whether to treat them or not. When the disease has existed four years at the time the diagnosis of latency is made, the patient's own defensive

used the first four injections may well be given on the first fifth tenth and fifteenth days, respectively. This will end the first two weeks of treatment and thereafter an injection should be given once each week until a total of twelve has been administered. In the last two weeks of this arsenic course the intramuscular injection of bismuth should begin the number of injections in this period depending upon whether a preparation suitable for weekly bi-weekly or triweekly administration is chosen. Overlapping of this sort is especially important in the first course because it probably prevents neurosyphilis. Then when the last of the arsenic is given the bismuth injections are continued alone until eight weeks of bismuth therapy have elapsed. During the last two weeks of this bismuth course weekly injections of arsenic should have been resumed and then the arsenic is continued alone until ten weekly injections have been given. Succeeding this and always overlapping two weeks at each end there will be another ten weeks of bismuth ten weeks of arsenic and a final ten weeks of bismuth. This point will be reached in about twelve months and if all has gone well the patient in whom treatment began in the seronegative primary stage may now be placed on probation. The seropositive primary or early secondary patient receives another ten weeks of arsenic ten weeks of bismuth ten weeks of arsenic and a final ten weeks of bismuth will be required to complete his approximately eighteen months of treatment. If bismarsen is being used of course no alternation can take place since arsenic and bismuth are combined in the one preparation the drug is merely injected weekly or perhaps better twice weekly if it can be borne without intermissions

until the total number of doses has been given.

It is better to end with a bismuth course than with an arsenical.

These standards, i. e., twelve months of continuous treatment for seronegative primary syphilis and eighteen months of continuous treatment for seropositive primary or early secondary syphilis, are based upon the correlation of clinical and serologic reports of the many patients favorably treated.

The Criteria of "Cure" If the patient has satisfactorily completed a standard treatment as outlined, he may be considered probably cured if he satisfies the following requirements:

1 Negative blood and spinal fluid at end of the treatment.

2 A full year of probation during which he receives no treatment, develops no lesions of syphilis and maintains a steadily negative blood the latter to be determined by serologic tests performed at frequent intervals but certainly no less often than every three months.

3 At the end of the year a complete examination, which must be negative for evidences of progress in the disease especially in the nervous system or cardiovascular apparatus, evidenced in the former by freedom from neurologic signs with a negative spinal fluid and in the latter by negative findings upon both physical and roentgenologic cardiovascular examinations.

4 Annual repetition of the complete examination as above except that if the second spinal fluid examination is negative, this test need not be routinely repeated in the subsequent years.

A physician can never guarantee that a patient is completely noninfectious; therefore all patients contemplating matrimony should be willing to undergo frequent physical and serological exam-

inations during the first ten years following infection. Certainly two years should have elapsed from the time of the infection even though the patient has had proper medication. After the lapse of five years only 30 per cent of the patients become infected and after the tenth year none.

Treatment Resistant Syphilis. The treatment resistant syphilitic is one in whom either spirochetes, lesions, or serologic manifestations, or all three are not affected by correct antisyphilitic therapy. The arsenicals are the drugs against which resistance is most often manifested, and most of the patients are early syphilitics, though resistance may be encountered in any stage of the disease. Many authorities feel that most of these resistant cases are seen in patients who have been treated with some form of combined therapy. There are, however, certain complications of syphilis which do not respond to any treatment. Deafness due to involvement of the auditory nerve, primary optic atrophy, perforating ulcers, and Charcot joints do not respond to antisyphilitic therapy. The percentage incidence of seroresistance is high in early involvement of the central nervous system, but in late syphilis seroresistance is a part of the disease and may be of favorable prognostic import.

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Every effort must be made to determine whether neurosyphilis, cardiovascular syphilis, hepatic, gastric, osseous, and other miscellaneous manifestations of late syphilis are present. If any of these manifestations of syphilis are discovered, the treatment is obvious; if none of them are discovered the question remains whether to treat them or not. When the disease has existed four years at the time the diagnosis of latency is made, the patient's own defensive

mechanism has already become firmly established. The Cooperative Clinical Group finds that the untreated cases will result as follows: (1) ultimate spontaneous "cure" (negative blood and spinal Wassermann, no lesions) 25 to 35 per cent of patients, (2) infection remains latent (positive blood but negative spinal fluid, no lesions) 25 to 35 per cent, (3) late cutaneous mucosal or osseous syphilis 10 to 15 per cent, (4) cardiovascular syphilis 10 to 15 per cent, (5) neurosyphilis 1 to 2 per cent, (6) other visceral syphilis, 1 per cent or less. This means that even if left absolutely untreated 50 to 70 per cent of the patients (always with the proviso that their syphilis is four years old when latency is diagnosed) will grow no worse and about half of that proportion will actually achieve spontaneous "cure" while about 20 to 30 per cent will develop visceral cardiovascular or some other form of late syphilis and 1 to 2 per cent will eventually have neurosyphilis.

By proper treatment the Group found it possible to achieve in 85 to 90 per cent of latent cases a result comparable to the spontaneous "cure" achieved in only 25 to 35 per cent of untreated cases. It is, therefore, evident that an appreciably greater proportion of individuals with true latent syphilis will get rid of their infection if correctly treated than if left untreated.

Treatment is reserved for those individuals in whom the most searching examinations have failed to detect any evidence of the disease other than the positive blood Wassermann reaction. Such patients are divisible into two groups: "early" latency with the full defense mechanism not yet established since the infection is less than four years old and "late" latency in which the

patient has had time to develop his resistance. In early latency treatment should be instituted for the reason that such patients being usually still quite young and sexually active, are a social menace through their still considerable likelihood to develop infectious relapse; furthermore, they probably received inadequate arsenical treatment and this means the likelihood of the occurrence of precocious tertiary syphilis, however in late latency the decision to treat or not to treat is more a matter of expediency. The studies of the Group reveal that the liability of a truly latent individual with a negative spinal fluid to develop neurosyphilis is extremely small. When ten years have elapsed since infection and no evidences of cardiovascular involvement are found when the diagnosis of true latency is made then such involvement is fairly certain not to occur later although no one can accurately appraise the normalcy of the small arteries of the brain, of the myocardium and the coronary system. It is the opinion of outstanding syphilologists that an individual who is over fifty years of age, whose infection is of long standing and gives no more evidence of its existence than the positive blood Wassermann, a fixed pupil or an absent knee jerk, should not be treated. Stokes believes that the patient in his thirties or forties may still have time to "use up" all of his immunity and he therefore advocates treatment in all these cases.

How to Treat. For "early" latency the best plan is to treat continuously for a year or arbitrarily for two years if Wassermann fastness occurs, and the amount of arsenic may well be curtailed in favor of bismuth. Many observers believe that the course should begin with bismuth to safeguard the patient against a Herxheimer reaction in a lesion whose

presence has not been revealed, treatment should also end with heavy metal. During one year treatment should consist of three bismuth courses of twelve weekly injections, each alternating with two arsenical courses of eight weekly injections each overlapping the drugs for one week at each end. Mapharsen is the arsenical to be preferred in latency because of its relatively lower toxicity. The course of therapy for latent syphilis is similar to that of early syphilis except that the first arsenical course is omitted and thus the bismuth course is lengthened. This plan is for early latency. In late latency the necessity for continuous treatment is not so imperative. Rest periods in these cases seem to do no harm.

Neurosyphilis

Neurosyphilis results from syphilitic invasion of the cerebrospinal system. This invasion occurs prior to and during the secondary stage. In those cases where treatment was started in the seronegative primary phase only about 0 per cent will develop neurosyphilis; in those cases starting treatment in the seropositive primary phase 21.8 per cent develop neurosyphilis, and in those cases starting treatment in the secondary stage 8.8 per cent develop neurosyphilis. In the relapsing cases of syphilis 35.9 per cent develop neurosyphilis. Neurosyphilis may be present many years before clinical signs are manifested. During this stage of so-called symptomatic neurosyphilis, the diagnosis can only be made by spinal fluid examinations. It is therefore of the utmost importance to resort to either lumbar puncture or cisternal (suboccipital) puncture in every syphilitic patient, regardless of the stage he happens to be in. In early syphilis, lumbar puncture should be performed

after six months of treatment and in late cases before starting treatment.

Primary acute syphilitic meningitis usually occurs in the early stages of syphilis, or results from its inadequate treatment. It is characterized by headache, pain and stiffness in the neck, nausea, and vomiting, mental signs, convulsions, cranial nerve palsies, aphasia, hemiplegia, increased cerebrospinal pressure and a positive spinal-fluid Wassermann reaction. It differs from other types of meningitis in its relative freedom from fever and its insidious onset.

Treatment. Headache can be relieved by resorting to lumbar puncture and removal of 30 to 50 cc. of cerebrospinal fluid every two to five days or until the headache is relieved; three lumbar punctures are usually required. Before performing lumbar puncture it is good practice to give a course of penicillin intramuscularly as already outlined or mapharsen or neosarsphenamine intravenously. Potassium iodide is given simultaneously in 5 to 8 gm. doses each day. Failure to respond to this treatment, of course necessitates resort to tryparsinamide, intraspinal or fever therapy.

The occurrence of such a syndrome as the above in the form of a true neurorecurrence is the "lighting up" of dormant neurosyphilis during a lapse in treatment will not be seen during the proper treatment of acute syphilis. Prolonged tertiaryism, which may involve the nervous system will also not be seen if the patient is being adequately treated.

Late neurosyphilis comprises paresis, taboparesis, tabes dorsalis, optic atrophy and meningovascular neurosyphilis, including such widely separated entities as transverse myelitis, syphilitic epilepsy, brain gumma, Charcot joint and late syphilitic hemiplegia.

All authorities are in agreement upon

the advisability of beginning treatment with a heavy metal preferably some form of bismuth. Iodide is hallowed by usage and is probably of value. When arsenic is added later it should be given as intensively as possible. The treatment advised is as follows: (1) Begin with a ten weeks course of bismuth injected weekly or perhaps preferably an eight weeks course using those preparations which may be safely injected twice or three per week. (2) Add arsenic in the last two weeks injecting every five days if possible for a course of ten injections. (3) The bismuth should overlap the arsenic again at the end or it may be possible to continue it in half dosage throughout the arsenic period. (4) Give three to five such courses (one round of bismuth and arsenic constituting a course) taking care to end with bismuth just as the beginning was made with bismuth. (5) Use iodides during all the courses of treatment.

The University of Chicago group (Walsh and Becker 1941) favor the following routine: five weeks of daily intramuscular injection of a soluble bismuth preparation together with daily intravenous administration of 25 to 100 cc. of 10 per cent sodium iodide solution (alternately weekly injections of insoluble bismuth and iodides daily by mouth) four weeks rest six weeks of arsenic and bismuth simultaneously four weeks rest eight weeks of bismuth four weeks rest six weeks of arsenic and bismuth four weeks rest eight weeks of bismuth four weeks rest six weeks of arsenic and bismuth.

As in latent syphilis, a lapse in treatment is not so serious a thing as it is in the treatment of early syphilis; however continuous treatment—no intermissions between courses—is preferable.

It is well to remember that the Herx-

heimer reaction with its typical intensification of symptoms, does occur with bismuth therapy.

Some cases of neurosyphilis can undoubtedly be arrested if not actually cured by one or the other of the types of treatment outlined. The largest available authoritative statistical analysis is that of Hopkins, who in 1933 reviewed the results in 1200 patients at the John Hopkins Hospital, with the following findings: (a) intensified routine antisyphilitic treatment was best in early neurosyphilis; (b) in paresis and labor paresis, malaria was preeminently the treatment of choice; (c) in tabes, malaria was best trypanamide almost as good and either one far superior to routine therapy; (d) in meningovascular cases, intraspinal therapy and trypanamide were superior to malaria treatment; (e) in optic atrophy subdural injections of arsphenaminized serum arrested the process in numerous cases in which it was advancing in spite of routine methods of treatment. Woods and Moore, of this same hospital definitely advised many years ago against trypanamide which they and many others consider contraindicated in optic atrophy.

More recently than the above Moore et al. (1938) have discussed the treatment of optic atrophy and reached conclusions which may be summarized as follows: (1) Untreated primary optic atrophy always becomes bilateral and practically always leads to permanent and complete blindness in seven years. (2) Adequate routine therapy delays the process and in an occasional case arrests it. (3) Subdural treatment arrests the process in about 60 per cent of instances, though it carries the risk of sudden loss of vision in about 10 per cent of those treated. (4) Malaria therapy brings about permanent arrest more frequently than

subdural therapy and therefore and be cause it is less dangerous to vision than subdural therapy it is the preferred initial form of treatment. (5) If visual failure progresses in spite of malarial subdural therapy should always be tried (6) Either of these forms of therapy should be followed by intensive routine antisyphilitic treatment

Bennett et al. (1939) find that in all types of active clinical neurosyphilis the simultaneous employment of fever therapy and chemotherapy is preferable to fever therapy alone. Goetto and Weinstein (1934) state that no type of antisyphilitic therapy affects deafness in late syphilis

Electric-Shock Therapy in Paresis
Reports on the use of electric-shock therapy in paresis indicate that this method has value. Petersen of the Rochester State Hospital, in one series obtained great improvement both clinically and in the spinal fluid of twenty patients out of thirty-four patients treated. The best response was obtained in the agitated group. The number of shocks administered in Petersen series varied from two to thirty-seven

Fever Therapy Since Wucherer's original introduction of malarial therapy in 1918, this original form of fever therapy has been extensively used throughout the world. Malarial fever therapy is performed by inoculation of the patient with the tertian or quartan malarial plasmodium. The plasmodium is taken from a patient who has undergone this treatment prior to having taken quinine. The injection may be given subcutaneously or intravenously. The period of incubation for malarial fever varies from eight to fourteen days; however if the malarial plasmodium is injected intravenously the incubation is greatly lowered, therefore the intravenous

method is preferable. About 2 cc. of blood is withdrawn from the donor and immediately injected into the recipient. The patient should not have more than eight paroxysms of fever excluding the prodromal fever. Only those paroxysms are recorded in which the temperature reaches 103° F or over.

The patient should be confined to bed immediately after the first elevation of temperature. Rectal temperature should be taken every three hours while the patient is awake until the beginning of a chill, then hourly until the temperature begins to drop. High temperature (106° F rectal) should not cause alarm; the high temperature is maintained for one hour. Should the temperature exceed 108° F a tepid bath should be ordered and repeated if necessary.

The tabetic symptoms are exaggerated by fever and, if too severe, they may be controlled by codeine and phenacetin. The blood pressure should be taken daily; the systolic and diastolic pressures always fall; the latter may fall to a proportionately lower level during the febrile period. The diastolic pressure fall need cause no concern; however if the systolic pressure falls below 90 supportive measures are indicated, and if it should fall below 70 the malarial fever therapy should be promptly stopped in order to prevent cardiac failure. A valuable sign indicating myocardial damage is tachycardia which continues during the afebrile period.

Red blood counts and hemoglobin determination should be made every three or four days. Should the hemoglobin fall below 50 per cent, the treatment should be stopped.

Nausea and vomiting may occur and if they prove uncontrollable, treatment is stopped and quinine dihydrochloride is given intravenously.

Whenever the paroxysms need be stopped thiolsimal is given and when the drug is stopped the malarial fever treatment continues as before. Induced malaria may be stopped by giving any of the various quinine compounds.

Later proposals to replace malaria with relapsing fever or rat bite fever did not meet with ultimate approval because these agents failed to prove their superiority; however recently the supremacy of malaria is being staunchly challenged by various physical methods of inducing fever: diathermy, short wave radiotherapy, conditioned hot air cabinets (hyperthermia), hot baths, and infrared radiation. Each of these new methods is being advocated almost with cultist fervor by its devotees, though the actual improvement which any of them effects over the classical malaria treatment is not proved.

A number of nonspecific chemical and biologic products have also been used: typhoid vaccine, gonococcus vaccine, milk sulfur, peptone, turpentine, sodium nuclemate, and blood (autohemotherapy). Of these the only one which has acquired any sort of lasting status is typhoid vaccine.

BLANKET WRAPPING. Epstein and Cohen (1935) have introduced this method but in doing so they stress the necessity of careful nursing attention throughout the treatment. No special preparation is necessary and the patient may have his breakfast. Wearing long woolen socks over the feet and legs, he gets into a bed on which have been placed first a large rubber sheet, then a large canvas sheet, and on top so that the patient will lie directly on it a heavy woolen blanket. As he lies on his back, each limb, the trunk, and the shoulders are wrapped individually with warm thin bath blankets. Then the entire body is

wrapped in a bath blanket, with only the face protruding; seven bath blankets having been used in all. Last, the heavy blanket, canvas sheet, and rubber sheet (extra large so as to cover the whole pile) are drawn around the patient, the whole bundle is wrapped in another heavy woolen blanket, and a last one is placed over the lower part of the body. The oral temperature and pulse at the temple are taken at half hour intervals and after each reading the patient takes through a tube 100 cc. of hot (100° F) lemonade containing 0.5 per cent sodium chloride and sufficient sugar to make it palatable. A temperature of 102° F (39° C) is usually reached in three or four hours, and 104° F (40° C) in five to six hours; it is maintained between 104° F (40° C) and 104.9° F (41.5° C) for the six hours which usually constitute a treatment. A rapid rise of temperature above this latter point should be controlled by loosening the blankets, fanning the body, administering cool drinks, and if the condition is alarming by a cool colonic flush. The pulse rarely exceeds 140 and the respiratory rate 28 per minute. A pulse rate above 180, a fall in systolic blood pressure below 80 mm of mercury, tendency to collapse, or the onset of tetanic indication for discontinuing the treatment. When the fever reaches its height restlessness may be controlled by hypodermic injection of morphine sulfate 0.015 gm ($\frac{1}{4}$ grain) and atropine sulfate 0.0004 gm ($\frac{1}{100}$ grain) after which the patient may sleep. Fever treatment is not given to patients of advanced age or to those with serious cardiac or pulmonary disease, advanced nephritis, extreme obesity, or a severe degree of arteriosclerosis, and the course of weekly treatments is discontinued during acute upper respiratory infection.

FEVER PRODUCED BY THE INTRAMUSCULAR INJECTION OF TRYPARSIN VACCINE: One cc of commercial tryparsin vaccine containing 1,000,000,000 organisms is diluted to 10 cc with physiologic saline solution and kept in the refrigerator for use on alternate days. Using a tuberculin syringe for accurate doses of 10,000,000

15,000,000 organisms are given for the first four or five treatments, and usually 10,000,000 or 20,000,000 for subsequent ones. The Nelson technique of giving a second injection at the height of the fever produced by the first is the one most usually employed. The first intramuscular injection is given in the morning. The patient usually experiencing a chill within thirty to ninety-two hours thereafter this lasts for a few minutes and

followed by a rise of temperature. When the rise is well established and progressing, the second intramuscular injection, using the same dosage is given. The interval between the two doses should not be less than two hours. Miller and Shaw (1933) found that a three- to six-hour interval is equally satisfactory provided the temperature has not dropped to nearly normal by this time. Temperatures of 103° F (40.56° C) to 107° F (41.67° C) are usually obtained by this method.

Fever therapy especially malaria is the treatment of choice for paresis with muscle atrophy in asymptomatic neurosyphilis and those types of neurosyphilis which do not respond to other forms of treatment.

Tryparsinoid. The average adult dose is about 5 gm dissolved in the ampoule in 10 cc of sterile distilled water and given at weekly intervals for eight to sixteen weeks, followed by a rest period of about six weeks. Many such courses are usually necessary to accomplish full results. The drug may also

be given intramuscularly or subcutaneously though it is often quite irritating under these conditions. Tryparsinamide solution is a pentavalent arsenical which is of great value in neurosyphilis. It should be injected as soon as it is prepared. It is usual to give biweekly during the rest period. Lorenz et al state that the results are better if 1 cc of mercury salicylate is given intramuscularly three days before each tryparsinamide injection. Some physicians are now giving full doses of either mercury or bismuth throughout the tryparsinamide courses.

Nearly all patients gain considerably in weight and well-being while taking the drug. Until recent years, the most frequent complaint was of ringing in the ears and a feeling of being dizzy and lured. Nitritoid reactions, nausea, vomiting and headache are of comparatively rare occurrence and urticarial reactions, dermatitis, and jaundice have been reported only a few times. It appears that tryparsinamide also occasionally activates mental and physical signs and symptoms in a most objectionable way.

As visual disturbances occur with sufficient frequency under tryparsinamide therapy the drug should be used only with the greatest care. Patients with preexisting optic involvement such as contracted fields or abnormal fundi, are more liable to injury than normal patients. Visual damage often occurs after the first few injections. If ten injections do not cause any visual damage, it is safe to continue the use of the drug. The most important signs of adverse action of the drug on the optic tract are subjective dimness of vision flickering, or shimmering sensations, or flashes of light also objective diminution in the visual acuity contraction of the visual fields, and changes in the fundi. The

occurrence of subjective symptoms should be thoroughly investigated for an objective basis. If no objective signs are found tryparsamide may be continued with caution. The presence of objective findings, as contraction of the visual fields is a contraindication to its further use.

The results with penicillin used alone are as yet inconclusive. However penicillin is of definite value in the treatment of all forms of neurosyphilis, and all patients should be given the benefit of this drug in the dosage already outlined with the highest dosage (10 000 000 units) given to patients with paresis and optic atrophy.

Congenital Syphilis

The diagnosis of early congenital syphilis rests on demonstration of spirochetes in the wall of the umbilical vein, a positive Wassermann development of osteochondritis and the presence of mucocutaneous lesions.

Demonstration of spirochetes in the wall of the umbilical veins definitely proves the diagnosis; however negative findings do not exclude congenital syphilis. The congenital syphilitic child is often premature. At birth it may be normal or it may be born extremely weak and manifest a syphilitic rhinitis (snuffles). These children often die within a few weeks. The eruption of congenital syphilis is rarely generalized; the sites of predilection are the palms, soles, and anogenital areas. Bullous lesions are often present on the palms and soles. Epiphyseitis may be present causing the child pain and limiting the movement of the extremity, a condition referred to as Parrot's pseudoparalysis.

The most important signs of late congenital syphilis are the following: interstitial keratitis, deafness, Hutchinson's

teeth, saddle nose, rhagades at angle of the mouth, frontal bosses, marked thickening of the inner third of the clavicle and Clutton's joint (symmetrical hydroarthrosis).

The blood Wassermann reaction is not always positive. Diagnosis of those whose Wassermann test is negative depends on objective symptoms. Physical examination and serological tests of parents should always be made.

Treatment in Congenital Syphilis

The principles of treatment of congenital syphilis are similar to the treatment given for late acquired syphilis. The early intensive and continuous method of treatment is absolutely necessary. It is principally during the first six months that the best hope of effecting a cure lies. A negative Wassermann reaction of the cord blood or of the mother's blood does not rule out the disease in the child. Unfortunately also a positive reaction of the cord blood or of the infant's own peripheral blood does not always mean syphilis. It has been convincingly shown on several occasions that these early reactions frequently become spontaneously negative and that the infants have not been actually infected however. Faber and Black (1936) have shown that the quantitative instead of the qualitative method of performing the Wassermann test could be used to considerable advantage since with it the decline of the test toward zero can be detected by the end of the first week or even sooner. Ingraham states that a single roentgenogram, made when the infant is six weeks old, will detect almost every case of syphilitic osteochondritis and periostitis.

Intravenous therapy in early congenital syphilis is difficult and often impossible; therefore the following therapy is the method of choice.

Week	Drug	Dose per mg. per Kilo- gram of Body Weight	Frequency of Administra- tion
1-2	bismuth salicylate	2	Every 3 days
3-10	sulfarsphenamine	10-15	Once daily
11-12	bismuth salicylate	2	Once weekly
13-23	sulfarsphenamine	15	Once weekly
24-26	bismuth salicylate	2	Once daily
27-46	sulfarsphenamine	15	Once weekly
47-51	bismuth salicylate	2	Once daily
52-61	sulfarsphenamine	15	Once daily
62-72	bismuth salicylate	2	Once daily
73-82	sulfarsphenamine	15	Once daily
83-84	bismuth salicylate	2	Once weekly

Bismuth salicylate in oil is the preparation of choice. It is administered in doses of 2 mg per kilogram of body weight. The dose must not exceed 22 mg per week.

Sulfarsphenamine is given intramuscularly in the gluteal region. It is a painless procedure. The initial dose is 10 mg per kilogram of body weight and subsequent doses should not exceed 20 mg per kilogram of body weight.

Mercury is indicated in the treatment for early congenital syphilis in cases of oak and undernourished infants where the above mentioned therapy might be dangerous. Thirty five per cent of U.S.P. mercury ointment in 1-gm. (15-grain) dosage is placed in the inner surface of the abdominal tunic and allowed to remain during the day. If the child shows signs of improvement following mercury the treatment as per above can be followed at the lapse of several weeks.

Stovarsol (acetarsone, spiroxol) is an active oral anti-syphilitic agent however its action is less rapid than that of bismuth and the arsenicals. It is usually

given for a period of nine weeks followed by a rest period of six weeks. The initial dose is 0.005 gm ($\frac{3}{125}$ grain) per kilogram (2.2 pounds) of body weight and during the last six weeks of the course 0.02 gm. per kilogram of body weight is given. The possibility of developing nephritis must be considered and the value of its use is problematical.

The treatment of late congenital syphilis is that of late or latent acquired syphilis. This stage of the disease is often extremely resistant to treatment, especially when its chief manifestation is interstitial keratitis, or some form of neurologic involvement. Late congenital syphilis differs from late acquired syphilis in that cerebrospinal syphilis and cardiovascular syphilitic manifestations are relatively rare in the former while interstitial keratitis is common in late congenital syphilis.

Continuous treatment is the method of choice in congenital syphilis. Mapharsen is administered in doses of 0.5 to 1 mg per kilogram of body weight—the maximum dose for a twelve year-old child should not exceed 0.03 gm. while in patients over fifteen years of age the adult dosage may be given.

Bismuth salicylate in oil (10 per cent suspension) is the drug of choice of the heavy metals. The dose is 2 mg per kilogram of body weight and the weekly dose should not exceed 100 mg.

A saturated solution of potassium iodide is of great value, especially in the treatment of interstitial keratitis, visceral involvement, and gummas.

Two years of treatment with alternating courses of mapharsen and bismuth is the accepted method.

Interstitial keratitis is a rather common complication of congenital syphilis. It often results in corneal scarring with partial or complete loss of vision. It

may involve both eyes and occasionally where only one eye is affected the other also becomes involved. Thirty six per cent of untreated cases of congenital syphilitics develop interstitial keratitis while only 2 per cent of those adequately treated develop it.

Sulzberger and Wolf recommend the following scheme of treatment for late congenital syphilis:

Five weekly injections of bismuth subarseniat
 Ten weekly injections of mapharsen
 Eight weekly injections of bismuth subarseniat
 Ten weekly injections of mapharsen
 Eight weekly injections of bismuth subarseniat
 Eight weekly injections of mapharsen

A blood test should be made prior to each arsenical course and the spinal fluid should be examined early in the course of treatment.

If the infant is ill with early congenital syphilis it may do badly under this routine. In such cases, it is considered advisable to begin the treatment with heavy metal and just attempt to prevent progression until the patient's general condition has improved. Howard (1930) considers it advisable to attempt some protection of the liver by injection of 50 to 100 cc of 10 per cent dextrose solution intravenously preliminary to each arsenical injection in the first course. Relapses occur in 3 to 10 per cent of the properly treated cases—in this event treatment should be continued. Fever therapy, protein shock therapy, large doses of riboflavin and vitamin A have also been advocated as worthy of a trial. Doses of penicillin comparable to those effective in adults are not entirely satisfactory in the treatment of infantile infection, according to Yampolsky and Herman (1946). Lentz and his coworkers (1946) state that "in the present state of our knowledge the treatment of infants with congenital

syphilis should be approached with extreme caution, reduced dosage and with emphasis on proper pediatric care. This is especially so for acute congenital syphilis. If penicillin is used the infant should receive about 150,000 units per kilogram of body weight in 120 equal divided doses, spaced every three hours around the clock for fifteen days. This therapy should be reinforced subsequently with a heavy metal as already outlined.

Syphilis in the third generation is rare. It is no contraindication to marriage. It is, however, advisable to treat the congenital syphilitic mother during pregnancy in order to prevent infection of the fetus in utero.

Treatment in Late (Tertiary) Syphilis

The typical forms of late syphilis of the liver—diffuse hepatitis, syphilitic cirrhosis, and hepatic gumma—call for the most caution in the choice of the anti-syphilitic drugs. A Herxheimer reaction in this organ may be fatal. Arsenical and bismuth therapy may produce such rapid healing and cicatrization that the portal circulation is obstructed with resultant ascites, or the bile ducts are occluded to such an extent that the most severe jaundice develops. Authorities are agreed that except in the rarest of instances the arsenicals and bismuth are absolutely contraindicated. Therapy should consist of the use of the iodides with mercury courses superimposed about as follows: iodides one week; iodides and mercury by mouth two weeks; iodides and mercury inunctions, eight or nine weeks; iodides alone for about four months then go through the cycle with mercury again and repeat the course if the patient's condition indicates further treatment.

In practically none of the other usual types of late syphilis—gastric and the rare intestinal and rectal forms of gastrointestinal syphilis; cutaneous and buccal manifestations of diverse nature; osseous and joint syphilis, etc.—need Herxheimer reaction be feared, treatment can, therefore, begin with the use of arsenic and can in general proceed much as in the treatment of acute syphilis. In the presence of a severe complicating nephritis, only the iodides can be used with full safety. In late syphilis of the bones and joints, surgical and orthopedic aid may be highly desirable; indeed, in Charcot joints accompanying tabes, the antisyphilitic attack is of itself impotent.

Penicillin is valuable in all forms of late visceral syphilis, but it is believed that preparatory therapy as noted above is the advisable procedure when it is used for hepatic syphilis.

Syphilis in Pregnancy

Syphilis in pregnancy is either early, latent, or late. Infection acquired shortly before or during pregnancy is altered by the pregnancy. The clinical signs of primary or secondary syphilis are minimized to such an extent that they may escape observation.

In early syphilis, the incidence of infantile congenital syphilis is about 90 per cent, while it is only 20 per cent in late latent syphilis. In untreated late latent syphilis, one obtains a history of miscarriages, stillbirth, congenital syphilitic offspring, and finally healthy nonsyphilitic children. The birth of a healthy child to a syphilitic mother with prolongation of the latent period is known as *late latent* law.

Treatment of early syphilis in the pregnant woman does not differ from the treatment of early syphilis in any patient. Antisyphilitic treatment should be given to all patients with latent syphilis regardless of pregnancy or the duration of the latent period. The prospective mother regardless of whether she has had adequate treatment or has been apparently cured, must be treated during each successive pregnancy.

Infection of the fetus occurs during or after the fifth month of pregnancy. Treatment started before the fifth month is prophylactic for the fetus, therefore, treatment of syphilis in the pregnant woman is solely for the purpose of safeguarding the fetus and giving birth to a healthy child.

Mapharsen is preferred in the treatment of syphilis in pregnancy because it is the least toxic of the arsenicals and does not produce nitritoid reaction. If treatment is started early alternating courses of mapharsen and bismuth subarsenate in oil are given continuously for the period of pregnancy. If treatment is not started until the seventh or eighth month, arsenic alone is given, although at more frequent intervals. If treatment is started later than bismuth in oil should be given simultaneously with the mapharsen. Lenz and his coworkers believe that penicillin has an excellent effect upon mother and fetus. The same dosages as advised for the nonpregnant patient are indicated.

After the child is born, the mother should have a complete examination, including lumbar puncture. Treatment of the mother is then started with whatever medication the stage of her disease might indicate.

TATTOOING

The term "tattooing" refers to the deliberate and permanent marking of the skin by the mechanical introduction of insoluble coloring matter the commonest being carmine indigo vermilion (red crystalline mercuric sulfide) cinnabar (sulfide ore of mercury) and carbon

Indian Indians, who applied figures of animals over their bodies to identify individual tribal clans

Religious designs were used by the early Anglo-Saxons The Council of Northumberland prohibited the practice of tattooing about A.D. 787

Tattoo figures are used by sailors to bring luck safeguard the sea journey and for protection from disease Some sailors believe that a good sized tattoo mark is as helpful as immunization against disease and assert that a well tattooed man is the most immune

Tattooing with mercuric sulfide (vermilion) for therapeutic purposes has been advocated in pruritus ani and port wine stains

Accidental Tattooing (Powder Stains) The material blown rubbed or injected into the skin may be carbon gunpowder (from explosions and especially in children from the careless use of firecrackers and guns) coal dust iron, gold bismuth and mercury The bluish black particles are of varying size and number and in these cases are embedded at various depths in the epidermis and even the dermis

Accidental tattooing may follow the application of certain iron compounds to the erosions of open varicellous lesions of dermatitis venenata The iron particles appear as blue-black marks

Accidental tattooing may follow extravascular injections of gold compounds (chrysiasis or chrysoderma) The pigmentation is local at the site of the injection However generalized pigmentation occurs also in some individuals who are receiving intravenous injections of gold and are subsequently exposed to much sunlight



Fig. 579: Accidental Tattooing Of the forehead. Embedded in the skin are many road particles which followed a tank explosion

They are introduced directly into the corium by means of needles Tattooing may however be accidental (powder stains)

Tattooing has been performed since the dawn of civilization The Syrians and Egyptians employed it centuries ago for treating lumbago sciatica and neuralgia Those of less-advanced civilization tattooed themselves to appease or discourage the evil spirits

Tattooing was practiced by the Amer-

Removal of Tattoo Marks The best procedure for removing deliberate tattoo marks is the so-called "French method." This consists in repeatedly tattooing (three or four times) the marked areas with a 40 per cent solution of tannic acid followed by rubbing the heavily punctured skin with a stick of silver nitrate. One may use the ordinary electric tattooing needle or a large flat cork, through which numerous needles have been thrust. The needles should project about 0.5 cm ($\frac{1}{4}$ inch). The area is thoroughly tattooed through the tannic acid solution; the stick of silver nitrate is then rubbed in thoroughly. A hard black, and adherent crust forms, which should be allowed to fall spontaneously. Glycerol of popond or glycerol of carond is injected into the tattoo. This treatment leads to solution of the tattooed tissue. Other methods in use include local applications of trichloroacetic acid, pure phenol, salicylic acid, sulfuric acid—1 gm. (15 grains) to 30 cc. (1 ounce) of water—concentrated nitric acid, 50 per cent solution of zinc chloride solution of mercury bichloride—0.5 gm. (10 grains) to 30 cc. (1 ounce) of water—cantharides plaster electrolysis, cutaneous trephine solid carbon dioxide and extension followed by skin-grafting. Small lesions are best excised.

The following peeling paste may facilitate removal

Resorcinol	40
Trichloroacetic	40
Glycerol q.s.	300
Use Massage into tattooed area twice each day	

Daily massage with *t. ble salt* until the formation of an erythema is another favorite treatment.

Large areas of tattoo marks are better left alone. Smaller ones are occasionally treated successfully.

Kromayer's Method for Removal of Poude Stains The instruments used are the rasp or the burr and small cylindrical knives or cutaneous trephine from 0.5 to 2 mm in diameter attached to a dental engine or to a dental drill mounted on a small portable motor similar to that used by engravers. If small areas are treated at a sitting, local anesthesia may be used. The whirling burr applied with astring pressure to the skin, will rasp off much of the pigment-containing epidermis. Heavy and deeper particles are then loosened with the cutaneous trephine and removed with forceps and scissors. The part treated is dusted with thymol iodid or powdered sulfathiazol and healing is practically complete within ten to fourteen days.

THROMBOANGITIS OBLITERANS

SYNONYMS *Buerger's disease* *preenile spontaneous gangrene*

Thromboangitis obliterans is a chronic recurring inflammatory disease chiefly of the peripheral arteries and veins, accompanied by thromboses which canalize and which are ultimately replaced by extensive fibrous connective tissue. This disease completely described by Buerger in 1908, was selected as a specific entity from a group of conditions somewhat

loosely known as "preenile gangrene."

Etiology The exciting cause is not definitely known. Ample evidence has been accumulated in recent years to indicate that all races are susceptible except perhaps the pure Negro. It is commonest in the Jew. Between 00 and 95 per cent of all cases occur among males. The majority of Buerger's cases

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Daily massage with table salt until the formation of an erythema is another favorite treatment.

Large areas of tattoo marks are better left alone. Smaller ones are occasionally treated successfully.

Kromayer's Method for Removal of Powder Signs. The instruments used are the rasp or the burr and small cylindrical knives or cutaneous trephine from 0.5 to 2 mm in diameter attached to a dental engine or to a dental drill mounted on a small portable motor similar to that used by engravers. If small areas are treated at a sitting, local anesthesia may be used. The whirling burr applied with varying pressure to the skin will rasp off much of the pigment-containing epidermis. Heavy and deeper particles are then loosened with the cutaneous trephine and removed with forceps and scissors. The part treated is dusted with thymol iodide or powdered sulf. thiazole and healing is practically complete within ten to fourteen days.

THROMBOANGITIS OBLITERANS

SYNOPSIS *Buerger disease* *prelesile gangrene* *gangrene*

Thromboangitis obliterans is a chronic recurring inflammatory disease, chiefly of the peripheral arteries and veins, accompanied by thromboses which calcify and which are ultimately replaced by extensive fibrous connective tissue. This disease, completely described by Buerger in 1908, was selected as a specific entity from a group of conditions somewhat

loosely known as *prelesile gangrene*.

Etiology. The exciting cause is not definitely known. Ample evidence has been accumulated in recent years to indicate that all races are susceptible, except perhaps the pure Negro. It is commonest in the Jew. Between 90 and 95 per cent of all cases occur among males. The majority of Buerger's cases

TATTOOING

The term "tattooing" refers to the deliberate and permanent marking of the skin by the mechanical introduction of insoluble coloring matter the commonest being carmine indigo vermilion (red crystalline mercuric sulfide) cinnabar (sulfide ore of mercury) and carbon

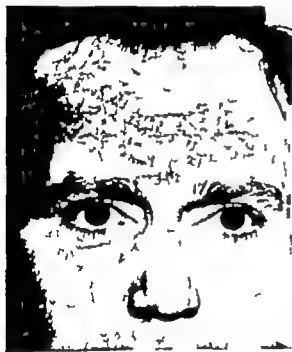


Fig. 579 Accidental Tattooing. Of the forehead. Embedded in the skin are many road tar particles which followed a tank explosion

They are introduced directly into the corium by means of needle. Tattooing may however be accidental (powder stains)

Tattooing has been performed since the dawn of civilization. The Syrians and Egyptians employed it centuries ago for treating lumbago sciatica, and neuralgia. Those of less-advanced civilization tattooed themselves to appease or discourage the evil spirits.

Tattooing was practiced by the Amer-

ican Indians, who applied figures of animals over their bodies to identify individual tribal clans.

Religious designs were used by the early Anglo-Saxons. The Council of Northumberland prohibited the practice of tattooing about A.D. 787.

Tattoo figures are used by sailors to bring luck safeguard the sea journey and for protection from disease. Some sailors believe that a good sized tattoo mark is as helpful as immunization against disease and a sign that a well-tattooed man is the most immune.

Tattooing with mercuric sulfide (vermilion) for therapeutic purposes has been advocated in pruritus ani and port wine stains.

Accidental Tattooing (Powder Stains) The material blown, rubbed, or injected into the skin may be carbon gunpowder (from explosions and, especially in children from the careless use of firecrackers and guns) coal dust iron, gold bismuth and mercury. The bluish black particles are of varying size and number and in these cases are embedded at various depths in the epidermis and even the dermis.

Accidental tattooing may follow the application of certain iron compound to the erosions of open vesicobullous lesions of dermatitis venenata. The iron particles appear as blue-black marks.

Accidental tattooing may follow extravascular injections of gold compounds (chrysiasis or chrysoderma). The pigmentation is local at the site of the injection. However generalized pigmentation occurs also in some individuals who are receiving intravenous injections of gold and are subsequently exposed to much sunlight.

Removal of Tattoo Marks The best procedure for removing deliberate tattoo marks is the so-called "French method." This consists in repeatedly tattooing (three or four times) the marked areas with a 40 per cent solution of tannic acid followed by rubbing the heavily punctured skin with a stick of silver nitrate. One may use the ordinary electric tattooing needle or a large flat cork through which numerous needles have been thrust. The needles should project about 0.5 cm. ($\frac{1}{4}$ inch). The area is thoroughly tattooed through the tannic acid solution, the stick of silver nitrate is then rubbed in thoroughly. A hard, black, and adherent crust forms, which should be allowed to fall spontaneously. Glycerol of popond or glycerol of caroid is injected into the tattoo. This treatment leads to solution of the tattooed tissue. Other methods in use include local applications of trichloroacetic acid, pure phenol, salicylic acid, sulfuric acid—1 gm. (15 grains) to 30 cc. (1 ounce) of water—concentrated nitric acid 50 per cent solution of zinc chloride solution of mercury bichloride—0.5 gm. (10 grains) to 30 cc. (1 ounce) of water—cantharides plaster electrolysis, cutaneous trephine, solid carbon dioxide and excision followed by skin-grafting. Small lesions are best excised.

The following peeling paste may facilitate removal

Resorcinols	40
Acid salicylic	10
Glycerine	500
100 Massage into tattooed part twice each day	

Daily massage with table salt until the formation of an erythema is another favorite treatment.

Large areas of tattoo marks are better left alone. Smaller ones are occasionally treated successfully.

Kronmayer's Method for Removal of Powder Stains The instruments used are the rasp or the burr and small cylindrical knives or cutaneous trephine from 0.5 to 2 mm in diameter attached to a dental engine or to a dental drill mounted on a small portable motor similar to that used by engravers. If small areas are treated at a sitting local anesthesia may be used. The whirling burr applied with varying pressure to the skin, will rasp off much of the pigment-containing epidermis. Heavy and deeper particles are then loosened with the cutaneous trephine and removed with forceps and scissors. The part treated is dusted with thymol iodide or powdered sulfathiazole and healing is practically complete within ten to fourteen days.

THROMBOANGITIS OBLITERANS

SYNONYMS *Buerger's disease; premenile spontaneous gangrene*

Thromboangitis obliterans is a chronic, recurring inflammatory disease chiefly of the peripheral arteries and veins, accompanied by thromboses which canalize and which are ultimately replaced by extensive fibrous connective tissue. This disease, completely described by Buerger in 1908, was selected as a specific entity from a group of conditions somewhat

loosely known as premenile gangrene.

Etiology The exciting cause is not definitely known. Ample evidence has been accumulated in recent years to indicate that all races are susceptible, except perhaps the pure Negro. It is commonest in the Jew. Between 90 and 95 per cent of all cases occur among males. The majority of Buerger's cases

were in the fifth decade of life. The general age tabulation is between the second and fourth decades especially between thirty and forty years of age. The diagnosis of thromboangitis obliterans should be regarded as improbable beyond the fifth decade. The ability of arterio-sclerotic disease to mimic thromboangitis obliterans is well known and indeed in some cases it is impossible to make a clear-cut differential diagnosis.

The predisposing causes are generally believed to be prolonged exposure to local trauma infection and excessive use of tobacco. One or more of these factors are found in most cases, but it is not uncommon to see patients in whom they are entirely absent.

There have been numerous instances where the thromboangitis obliterans symptom complex has been seen in association with known specific diseases, such as typhus fever and other rickettsial infections.

Vascular Pathology Buerger summarizes the morbid anatomy as a simultaneous inflammation of all the vascular layers, quickly followed by red thrombosis. The clot then undergoes organization and partial canalization and capillarization together with the formation of foci of giant cells. The normal architecture of the vessel wall is usually preserved. Beyond this point further replacement with fibrous connective tissue involves not only the arteries but also the accompanying veins, and even nerves, until they become a fibrous cord.

Because of the recurring nature of thromboangitis obliterans, it is possible to find various stages of the disease along the course of an affected artery or vein. There is some difference of opinion, however as to what constitutes the vascular changes of thromboangitis obliterans.

Intimal proliferation occurs in thromboangitis obliterans but it seems to be almost as prominent in arteriosclerosis obliterans. Thus, a patient with thromboangitis obliterans in the fourth decade may have some arteriosclerotic changes, upon which are superimposed the pathological alterations of thromboangitis obliterans. It is probably nearer the truth to say that there is no typical histopathological picture in thromboangitis obliterans. In practice, it is often necessary to examine numerous tissue specimens before a definite diagnosis can be made. Not infrequently the pathologist reports that the tissue pathology suggests "thromboangitis obliterans or is compatible with the diagnosis."

Abnormal Physiology In association with the pathological changes in the major vessels, there is also arteriospasm in communicating arteries and arterioles. While there is no absolute proof of this, it can safely be inferred, since some patients exhibiting very low skin temperature particularly early in the disease are greatly benefited by paravertebral block or fever therapy. It is important to evaluate the degree of arteriospasm present for here we can do the greatest good for the patient.

The only important pathologic change produced by this disease is diminution of flow in the peripheral circulation.

Because of the inability of the peripheral circulation to maintain adequate blood supply to the extremities during muscular activity we have the basis for one of the most reliable symptoms in peripheral vascular disease. This symptom is known as "intermittent claudication." It is important to establish criteria for it, and these are

1. Marked fatigue or cramping pain recurring in the foot or calf muscles or both

2. Always brought on by a definite amount of exercise

3. Always relieved by simply stopping the exercise. If the exercise is walking, the patient need not sit or lie down, he simply stands still until the discomfort leaves him, whereupon he starts to walk again for the usual distance only to stop again.

Symptoms The symptoms depend upon the degree and position of vascular occlusion, and are modified by the frequently associated arteriospasm and thrombophlebitis.

The symptoms usually develop insidiously but may occasionally be fulminant. The fulminating type is the exception. Occasionally a patient is incapacitated by the disease in a few weeks, and amputation is required in two or three months. Most often however cases with fulminant onset seem to stabilize themselves after a few weeks of hospital care, and are much the same as the cases with an insidious onset.

Migratory thrombophlebitis usually does not cause much suffering. The thromboses are usually found in the course of the internal saphenous vein between the ankle and the knee joint. They are small and quite tender. During the acute stage the thrombus is surrounded by a overlying cutaneous zone of erythema. As the lesions become less tender the erythematous zone surrounding them becomes brownish, and then progressively lighter until it is no longer visible.

It is a rare patient who presents himself with migratory phlebitis, for this system complex is usually seen in combination with other more disabling complaints.

Coldness of the extremity to a degree that it is unpleasant is common. Examination at this time will usually reveal

signs of arterial insufficiency. The dorsalis pedis artery and the posterior tibial artery in the foot may both be occluded.

The skin of the foot may vary from a cadaveric hue to a purplish red. If the foot is deep red in the horizontal position, the color will become deeper and cyanotic when the extremity is lowered. If the extremity is elevated to a 45-degree angle or more the foot will blanch considerably, often becoming chalk white, but the deep red returns



Fig. 580 Thromboangitis Obliterans. Note the three trophic ulcers over the instep and the loss of subcutaneous fat over the toes and the foot in general.

as soon as the extremity is lowered. If the examiner places himself so that he may view these changes of skin color on the plantar surface they are all the more striking.

In the early phases of the disease, there is frequently a moderate amount of edema about the toes and dorsum of the foot. This is probably caused by increased capillary permeability asso-

ciated with anoxia. Bleb-formation is commonly seen at this time.

After a few weeks, the arterial circulation tends to compensate by canalization of the thrombus and the enlargement of normally small collateral vessels. In the favorable cases and with rest pain disappears. Tissue edema becomes markedly decreased and may disappear. The skin may now be similar in appearance to that seen in early dry gangrene. There is marked wrinkling and dryness, with loss of subcutaneous fat. The loss of muscle tissue around the toes makes the phalangeal joints more conspicuous. The skin may become parchmentlike and limit the motion of the phalangeal joints.

Changes in the retinal vessels are stated to be common in the late stages of the disease.

Diagnosis. Thromboangutis obliterans is a clinical entity. There is no test or sign however that establishes the diagnosis beyond reasonable doubt. It is necessary to obtain a history as carefully as though the symptoms were in the peritoneal or pleural cavity. It is estimated that more than 50 per cent of the cases with peripheral vascular disease can be diagnosed from the history alone.

The typical patient is a young white male with a history or evidence of migratory thrombophlebitis, complaining of pain in an extremity brought on by a specific amount of exercise and relieved by standing rest. Examination shows loss of one or more of the peripheral arterial pulses, with coldness and color changes in the involved member. More often however there is no history or evidence of migratory thrombophlebitis. When thromboses occur distal to the posterior tibial artery with a pulsating dorsalis pedis and posterior tibial ar-

tery the diagnosis becomes difficult. It may be possible to make a diagnosis early in atypical cases from subsequent events.

The Pachon Oscillometer. In addition to the physical examination, there are more precise methods of investigation. The Pachon oscillometer is a useful instrument. It measures the distention of the artery following systole of the heart. If the sensitivity of the instrument is examined by lightly tapping the inflated cuff it will be seen that it takes a considerable impulse to register at all. For this reason it is extremely difficult to evaluate oscillometric readings between zero and one-half unit. A zero oscillometric reading at the ankle may or may not mean a complete occlusion at that point. At the level of the knee a zero oscillometric reading is extremely serious, and most often means a poor prognosis. An oscillometric oscillation of one-half unit or more at the ankle is within normal limits.

In thromboangutis obliterans, the oscillometer will indicate severe arterial damage in the involved extremity while readings in the uninvolved member will be quite normal. This is not the case in arteriosclerosis obliterans where the arterial disease is bilateral and frequently the degree of involvement is symmetrical or nearly so. This observation constitutes the most important finding in the differential diagnosis between thromboangutis obliterans and arteriosclerosis obliterans.

The Histamine Flare Test. This indicates the efficiency of the cutaneous circulation. It may be used as a test of the collateral circulation when the major arteries are known to be occluded. The test may be performed at any level on the extremities, and is widely used to determine the lowest level of amputation.

when that procedure becomes unavoidable. In the performance of this test a solution of histamine phosphate (1:1000) is used. About 0.5 cc. of the histamine solution is drawn up in a hypodermic syringe. The skin area to be tested is wiped gently with alcohol, which is allowed to dry by evaporation. One drop of the histamine solution is injected intradermally at the site to be tested. In normal skin, the reaction to the histamine occurs within 5 to 10 minutes, and consists of a zone of erythema at the site of the injection. In the center of this zone is a slightly elevated wheal. It is sometimes easier to feel the wheal than it is to see it. Occasionally only half the reaction occurs, and either the wheal or the flare may be absent. If neither wheal nor flare appears within 10 minutes, there is severe circulatory impairment. It is well to test the histamine solution on one's own hand to be certain that it is potent.

Determination of Skin Temperature. Another instrument of precision in general use is the thermocouple. It is used to determine skin temperature. Practically it is chiefly used to determine the actual rise in skin temperature following para-arterial block or reflex vasodilatation. With a little practice it is possible to determine differences as little as 0.1° by palpation with the back of the hand; this is sufficiently accurate for practical purposes. In examining patients by this method, it is important that the examiner's hand should not be too warm. Differences in temperature are more readily determined if the temperature of the examiner's hands is about the same or lower than the skin of the patient's extremities.

If sympathectomy is contemplated, it is necessary to obtain accurate skin temperatures. The patient is disturbed com-

pletely and covered with a cotton sheet; the examining room temperature should be in the neighborhood of 70° F. The patient should be on a table or bed for at least thirty minutes and encouraged to relax as much as possible. Following para-arterial block, a rise in skin temperature of the extremities as little as 5° F. should lead one to contemplate sympathectomy. The newer methods of approach have greatly increased the value of this procedure.

Differential Diagnosis. The only other disease which may resemble thromboangitis obliterans in almost every detail is arteriosclerosis obliterans. Indeed, it may be impossible to differentiate these diseases after careful study and sometimes even after amputation and histological study of occluded vessels.

In arteriosclerosis the patient is usually in the fifth decade of life, or older; the duration of the complaint is frequently a matter of years before advice is sought, and the involvement is bilateral and progresses very slowly. If sufficient calcium is deposited in the arterial vessels, x-ray examination may indicate this calcification.

The vasospastic states including Raynaud's disease occur in females predominantly, are worse in cold weather, affect the upper extremities more frequently and occur in attacks which are distributed symmetrically. Gangrene is rare, and when it does occur is superficial. Characteristic changes in the bones of the terminal phalanges are easily demonstrated by x-ray.

Treatment. This phase of the disease leaves much to be desired. The patient is best treated in a hospital at least during the acute stages of the disease. If gangrene is present the patient should not be allowed bathroom privileges. The extremity should be kept dry and clean

Ointments and wet dressings tend to macerate the involved area, and almost invariably make matters worse. The use of strong antiseptics and keratolytics is especially contraindicated. If edema is present the extremity should be elevated slightly (10 to 20 degrees) preferably on pillows, the heel kept free of the pillows or bed. If no edema is present the foot should be at body level or even lowered by the use of pillows placed under the hip and shoulders. A cradle with one or two 25 watt bulbs, is beneficial. The foot should never be heated above the patient's tolerance. A small piece of sterile gauze inserted between the toes to keep them separated is frequently useful. In nongangrenous areas where the skin is dry a small amount of lanolin makes the skin more pliable.

Procedures in Pain at Rest. Considerable pain at rest presents a formidable problem unless the situation is corrected; the patient usually gets worse rapidly. The inability to rest and maintain an adequate food intake because of constant pain contributes in no small measure to the poor result in these cases. The use of *intravenous typhoid vaccine* (Type II) beginning with 10,000,000 organisms every other day occasionally relieves this most disturbing symptom. The *crushing of terminal nerves* in whose distribution the pain is felt is a useful but limited procedure. The incision of course, should be made well out of the area of tissue whose blood supply is diminished. *Paravertebral block anesthesia* may relieve pain even though it has little effect on the skin temperature. This procedure is quite safe in skilled hands and if it gives relief it may be repeated frequently without untoward effect.

Use of Suction and Pressure. Since all treatment is directed toward preventing irreversible tissue damage and until

canalization of the occlusion and development of a collateral circulation occurs, it is important to try the means at one's disposal as early as possible.

The *suction and pressure treatment* is sometimes useful. Its use is contraindicated in the presence of gangrene, especially if there is infection. The *positive pressure cuff* if it can be tolerated by the patient, sometimes relieves pain and is worthy of trial. Neither of these methods is of benefit during the acute phases of the disease, but rather finds its greatest usefulness during the recovery phase of the disease.

Precautions and General Measures. The use of tobacco in any form should be denied every patient who has *thromboangitis obliterans*. Preparations containing iodine merthiolate sulfonaphthol phenol (carbolic acid) cresol or saponated solution of cresol should be avoided. *Trophicosis* should be treated but not with preparations containing salicylic acid. A safe method of treatment is to soak the feet for half an hour twice daily in a 1:8000 solution of *potassium permanganate*. *Harm Sitz baths* with water as warm as can be tolerated comfortably may be used for periods of ten to twenty minutes one to three times each day. Electric pads or hot water bottles should never be applied directly to the skin.

Postural exercises may be helpful. The patient should elevate the extremities for three minutes, then place them in the dependent position for three minutes, and then lie with them in a horizontal position for three minutes. These procedures should be repeated five times, two or three times a day. Some authors feel that a diet high in calcium or the addition of half a teaspoonful of calcium lactate or a heaping teaspoonful of calcium gluconate is advisable an hour be-

fore meals. Injections of 250 cc. of a 2 per cent solution of *sod m citrat* as often as every day or as infrequently as two or three times a week for a considerable period may help.

Fever Therapy: One of the best methods of increasing the circulation in thromboangitis obliterans is the repeated induction of fever by means of intravenous injection of typhoid vaccine. For the convenience of administration, a stock solution should be diluted with an isotonic solution of sodium chloride so that 1 cc. contains 100 000 000 killed bacteria. The desired reaction is elevation of the oral temperature to from 101 to 102 F. Chill, headache and malaise may result. Excessive elevation of the oral temperature, severe chills, and serious malaise are not necessary and are to be avoided if possible. The initial injection contains from 15 000 000 to 25 000 000 bacteria for a man and from 10 000 000 to 15 000 000 for a woman. Ordinarily each subsequent dose is increased by from 15 000 000 to 25 000 000 bacteria, but the amount injected depends on the reactions. If the reaction to any injection is too severe, the subsequent injection should be increased only slightly or not increased at all. If the reaction is too mild, the next injection should contain from 25 000 000 to 50 000 000 bacteria more than the preceding one did. Injections may be given once or twice a week for six or eight weeks, after which a rest period of several weeks is advisable before another course is begun. Contraindications to the intravenous injection of typhoid vaccine are advanced debilitating diseases, diseases of the coronary and cerebral arteries, chronic nephritis, myocardial insufficiency, active pleurisy or pericarditis, latent or quiescent appendicitis, and cholecystitis.

A very recent advance in the treatment of these patients is the use of *tetraethyl-ammonium chloride*. This drug apparently produces its effect at the ganglia of the autonomic nervous system. Since vasomotor tone is a function of the sympathetic nervous system alone, this substance produces a peripheral vasodilatation that equals the effect of regional sympathetic ganglion block with procaine. The drug is most effective intravenously in doses of from 1 to 5 cc. diluted in isotonic saline. The effect occurs within twenty seconds and begins as a metallic taste in the mouth. This is quickly followed by a slight sensation of numbness, coldness, and finally tingling in the extremities. Excessive sweating ceases completely. The patient then notices that the hands and feet are warm. This last effect is striking in patients who have cold hands and feet. Among other effects, there is loss of accommodation which interferes with near vision. The pupils are partially dilated and respond sluggishly to light. There is a considerable fall in blood pressure in hypertensives followed by a short period of postural hypertension. Obviously this drug is a formidable addition to the drug armamentarium in peripheral vascular disease. Untoward effects of the drug may be stopped immediately by injection of a therapeutic dose of epinephrine. Moore, G. K., Rennie, et al. have reported the use of this substance in cases where vasoconstrictor tone was believed to be a factor. In arteriospasm associated with thromboangitis obliterans, the spasm has been controlled quickly relieving rest pain and, in ambulatory patients, prolonging exercise tolerance in the presence of intermittent claudication. The author's experience with this drug has been too short to permit a complete evaluation. Enough cases have been studied, however to permit an optimistic attitude

with regard to tetra-ethyl-ammonium chloride

Conservative and Surgical Measures in Gangrene With the present-day use of the sulfonamides and penicillin infection is rarely an indication for radical surgery. When gangrene occurs it is proper to wait until demarcation is clearly evident before surgical intervention. If the gangrene involves only the toes, it is usually better to wait until spontaneous amputation is well developed so that it is possible to achieve removal of the necrotic tissue with as little surgical interference as possible. In some instances *débridement* may be advisable for the relief of pain. This is

satisfactory if the area surrounding the necrotic tissue has a fairly good circulation. It is only rarely that a toe can be successfully amputated, for the incision ordinarily does not heal. When fingers are amputated on the contrary the wounds almost invariably heal. Other measures, such as those mentioned in the preceding paragraphs should be used. When massive gangrene affects an entire foot, amputation is ordinarily the best solution. In about 80 per cent of the cases, healing follows amputation of the extremity below the knee but it is occasionally necessary to perform reamputation above the knee. Necessity for amputation of the forearm has never been observed.

TINEA

Tinea Barbae

SYNONYMS: *Tinea sycosis*, *beard's itch*, *parasitic sycosis*, *tinea trichophytina barbae*, *ringworm of the beard*, *hyphomycotic sycosis*, *trichophytosis barbae*

Tinea sycosis is a chronic inflammatory disease involving the bearded region and is characterized by papules, pustules, tumorlike swellings associated with varying degrees of hair destruction.

Varieties Two varieties occur the superficial form and the deep variety the so-called "sycosis parasitaria."

Incidence It involves the bearded region in adult males. Farmers or those coming in contact with infected animals are especially liable to infection.

Etiology The superficial type of *tinea trichophytina barbae* is usually caused by the endothrix variety of the *Trichophyton* fungus. Involvement of the beard by the microsporon is extremely rare.

Pathology The superficial type is characterized by the presence of round

oval or inflamed and slightly infiltrated areas covered with scales, vesicles, or pustules. They progress by peripheral extension and show a tendency to heal in the center. In the deep variety the fungus enters the hair follicle and causes folliculitis and perifolliculitis characterized by hard slowly developing chronically inflamed nodules which have a tendency to ulcerate and discharge mucopurulent or serosanguineous material. Hairs extracted from these areas appear whitish and succulent at the roots and the parasites can be readily demonstrated after the hairs have been treated with a heated 10 to 30 per cent solution of sodium hydroxide.

Symptoms The superficial as well as the deep variety begins as superficial scaling patches and their clinical courses parallel fairly closely the depth and extent to which the hairs become involved. The hairs affected are generally loose, dry and brittle and when extracted the bulb appears large and white due to adherent detritus. Frequently

there is alopecia in the diseased patches. The deep type of the disease develops slowly and produces nodular thickenings and kerionlike swellings. As a rule these are confluent and form diffuse boggy infiltrations with abscesses. The underlying skin is inflamed and the hairs are loose or absent and pus may be



Fig. 581 Tinea Barbae or Sycoosis Parasitica. (Courtesy of Dr. Ralph Bernstein.)

expressed through the remaining follicular openings. Generally the lesions are limited to one part of the face or neck in male adults, although occasionally they are widespread over the bearded region. The usual locations are the bearded parts of the neck or of one cheek. As a rule the upper lip escapes involvement.

The trichophyton test is apt to be positive.

Diagnosis. Tinea barbae is differentiated from *sycoosis vulgaris* by the occurrence of the mandibular or submaxillary areas, while *sycoosis vulgaris* usually in-

volves the upper lip and the bearded region. The characteristic lesions of *sycoosis vulgaris* are superficial hair pierced pustules or papules. Parasitic *sycoosis* produces either superficial circinate, scaling patches, or deep nodules which are circumscribed swollen patches within which some of the hairs are loose or broken off. In *seborrheic dermatitis* and *contact dermatitis* the process is superficial and the hair shafts are not involved. Tertiary *syphilis* produces tissue destruction followed by atrophic scarring. Tinea barbae is differentiated from *actinomycosis* by cultural and microscopic examination which reveals either fungi or actinomycetes.

Prognosis. Several months of treatment usually results in a cure. Relapses are not uncommon.

Treatment. If the involved area is crusted, soap and water or poultices of starch and boric acid are indicated. Manual epilation followed by bathing twice daily with hot Fleming's solution diluted with 10 parts of water is helpful where the involved areas are not very extensive.

Wet packs of hot solution of 1:10,000 bichloride of mercury, zephiran chloride (1:1000) or potassium permanganate 1:8000 twice daily are valuable adjuncts to treatment, and should be followed by rubbing with 30 per cent ammoniated mercury ointment or 5 per cent ointment of sulfadiazine. Manual epilation is helpful. Gradually ascending doses of trichophyton have been used with benefit.

Cases which do not respond to this treatment should receive roentgen therapy. Fractional roentgen dosage rather than full doses is perhaps the safe way to effect epilation. The suggested dosage is 80 r (1½ E.D.) given through 3 mm. aluminum at two-day intervals for a total of five treatments. A second

exposure of x ray should not be given for at least two months. In resistant cases Mook has used *fever therapy* (intravenous typhoid vaccine on three successive days).

Andrews suggests that the face should be disinfected after epilation. He suggests either *Schlumsky's solution* (phenol 30 parts camphor 60 parts alcohol 10 parts) or a solution of *bichloride of mercury* 1:1000 in fluid extract of witch hazel.

Tinea Capitis

SYNONYMS: Ringworm of the scalp, *he pes tonsurans*, *tinea trichophytina capitis*, *tinea tonsurans*

Tinea capitis is an inflammatory contagious disease of the scalp occurring usually in school children and only occasionally in infants and adults.

Varieties The different types of *tinea tonsurans* produce varying clinical pictures. The *Microsporon audouinii* (Cruby) and the *Microsporon lanosum* are varieties of the genus *Microsporon*; the former is referred to as the human type while the latter is referred to as the animal type. The *Microsporon audouinii* is the most common and produces most of the cases of *tinea capitis*. It is transferred from human to human and it is known that school children are often carriers despite clinical absence of signs. It is the causative organism in epidemic ringworm. The *Microsporon lanosum* is commonly found in cats, dogs and horses, and only occasionally produces *tinea tonsurans* in children. Infection with *Microsporon lanosum* is self limited.

Tinea tonsurans trichophytica, which is in the minority of cases of *tinea tonsurans*, is produced by the *endothrix* or the *ectothrix*. Source of infection of *Trichophyton gypsum* is infected cattle. It is apt to be more acute than Micro-

sporon lanosum infection and is also self limited.

Incidence *Tinea tonsurans microspora* is almost exclusively a disease of children. *Tinea tonsurans trichophytica* is also a disease of childhood although it occasionally occurs in infants and adults.

Etiology *Tinea tonsurans* is caused by any one of the several varieties of ringworm fungus. Both the *Microsporon* and the *Trichophyton* group of fungi have species which are chiefly pathogenic for lower animals and species which are



Fig. 562 Tinea Tonsurans.

chiefly pathogenic for humans. Infections contracted from animals are but lightly contagious among humans, and when contracted by humans are self limited and respond more or less promptly to topical therapy. Their self limitation is due to the local inflammatory reaction they induce and to the development of antibodies. The *Microsporon lanosum* usually produces but slight inflammatory reaction. They are nonepidemic and also self-limited.

The backs and headrests of chairs in motion picture theaters appear to be a potent source of transmission of microsporiasis. Schwartz and his collaborators believe the greatest source of transmis-

as perfolliculitis. Hairs extracted from these lesions appear whitish and succulent at the roots, and the parasite can be easily demonstrated after being treated with sodium hydroxide. The



Fig. 583. Tinea Tonsurans. Caused by *Microsporum lanosum*.

sion of epidemic ringworm to be the barber shop (combs, brushes, scissors, and clippers).

Pathology The small-spored fungus causes no marked degree of disease. In the ectothrix and the end thrix varieties, of *Trichophyton* the fungus enters the hair follicle and causes folliculitis as well

as perfolliculitis. The primary lesion of *tinea tonsurans* is a macule. The macule is sharply circumscribed and covered with grayish or whitish scales and with

fungi occur within the hair and in the adjacent epithelium of the follicle. The fungi never invade the connective tissue of the corium.

exposure of x ray should not be given for at least two months. In resistant cases Mook has used *fever therapy* (intravenous typhoid vaccine on three successive days)

Andrews suggests that the face should be disinfected after epilation. He suggests either *Schlumsky's solution* (phenol 30 parts camphor 60 parts alcohol 10 parts) or a solution of *bichloride of mercury* 1/1000 in fluid extract of witch hazel

Tinea Capitis

SYNONYMS: *Ringworm of the scalp, he pes tonsurae, tinea trichophytina capitis, tinea tonsurae.*

Tinea capitis is an inflammatory contagious disease of the scalp occurring usually in school children and only occasionally in infants and adults

Varieties The different types of *tinea tonsurans* produce varying clinical pictures. The *Microsporon audouinii* (Cruby) and the *Microsporon lanosum* are varieties of the genus *Microsporon*; the former is referred to as the human type while the latter is referred to as the animal type. The *Microsporon audouinii* is the most common and produces most of the cases of *tinea capitis*. It is transferred from human to human, and it is known that school children are often carriers despite clinical absence of signs. It is the causative organism in epidemic ringworm. The *Microsporon lanosum* is commonly found in cats, dogs, and horses, and only occasionally produces *tinea tonsurans* in children. Infection with *Microsporon lanosum* is self limited.

Tinea tonsurans trichophytica which is in the minority of cases of *tinea tonsurans*, is produced by the endothrix or the ectothrix. Source of infection of *Trichophyton gypsum* is infected cattle. It is apt to be more acute than *Micro-*

sporon lanosum infection and is also self limited.

Incidence *Tinea tonsurans microsporiga* is almost exclusively a disease of children. *Tinea tonsurans trichophytica* is also a disease of childhood, although it occasionally occurs in infants and adults.

Etiology *Tinea tonsurans* is caused by any one of the several varieties of ringworm fungus. Both the *Microsporon* and the *Trichophyton* group of fungi have species which are chiefly pathogenic for lower animals and species which are



Fig 582: Tinea Tonsurans.

chiefly pathogenic for humans. Infections contracted from animals are but slightly contagious among humans, and when contracted by humans are self limited and respond more or less promptly to topical therapy. Their self limitation is due to the local inflammatory reaction they induce and to the development of antibodies. The *Microsporon lanosum* usually produces but slight inflammatory reaction. They are nonepidemic and also self limited.

The backs and headrests of chairs in motion picture theaters appear to be a potent source of transmission of microsporous. Schwartz and his collaborators believe the greatest source of transmis-

as perfolliculitis. Hairs extracted from these lesions appear whitish and succulent at the roots, and the parasite can be easily demonstrated after being treated with sodium hydroxide. The



FIG. 583. Tinea Tonsurans. Caused by *Microsporum lanosum*.

ions of epidemic ringworm to be the barber shop (combs, brushes, scissors, and clippers).

Pathology The small-spored fungus causes no marked degree of disease. In the ectothrix and the endothrix varieties, of *Trichophyton*, the fungus enters the hair follicle and causes folliculitis as well

as perfolliculitis. Hairs extracted from these lesions appear whitish and succulent at the roots, and the parasite can be easily demonstrated after being treated with sodium hydroxide. The

Symptoms The primary lesion of tinea tonsurans is a macule. The macule is sharply circumscribed and covered with grayish or whitish scales and with

borders that are slightly elevated above the plane of the norm. Tendency for central clearing may be present. After a variable time invasion of the hair and hair follicles occurs. The fungi gradually spread into the follicle and then spread in an upward direction beneath the cuticle stripping it off they also



Fig. 584. *Tinea Tonsurans*.

invade a short distance downward and form the so-called "fringe of Adamson." The fungus, however, never invades the hair bulb nor its vascular papillae.

The *Microsporon audouinii* (the human type of fungus) is the commonest cause of tinea tonsurans. This type of fungus first invades the superficial portion of the scalp epidermis and then attacks the hair. The earlier lesions resemble patches of tinea circinata. These patches are circinate or oval, are sharply margined and occur more often on the vertex and parietal areas of the skull. The hairs within the patches are broken off at irregular distances from the surface. The hairs have a gnawed-off appearance, are

lusterless, break off easily and can be painlessly epilated. The surface of the scalp is covered with fine, dry, grayish white scales. A slightly erythematous border on which the hairs are shorter than in the center surrounds the patch. Microscopic examination of the epilated hair from the patch shows it to be surrounded by a whitish sheath which extends from 1 to 3 mm above its exit from the follicle. The sheath consists of a thick layer of spores mosaically arranged. The free end of the hair has a brushlike appearance (trichorrhexis).

If tinea tonsurans should be caused by the *Microsporon lanosum* (the animal type) the lesions show more inflammatory signs.

Infection of the scalp with the *Trichophyton* fungus also occurs. The fungus is termed "ectothrix" (*M. lanosum*, *M. audouinii*, *T. gypseum*) when the spores are arranged in a sheath around the hair; it is termed "endothrix" when the spores invade the shaft (*Trichophyton* of human origin) and "ectothrix" when present as a sheath and within the shaft.

The species of *Trichophyton* are usually divided into two main groups: the endothrix and ectothrix. The endothrix variety is found almost exclusively in man and grows only within the hair substance. The ectothrix variety is of animal origin, common to dogs, cats, cattle, horses, etc. It not only invades the hair substance but also invades hairy surfaces.

The *Trichophyton* endothrix type of tinea tonsurans begins with invasion of the epidermis of the scalp, which is soon followed by involvement of the hair; the fungus filling the interior of the hairs. The patches usually resemble those caused by the *Microsporon* but the patches are generally smaller, the alopecia less complete, and the hairs

broken off at the surface of the scalp. Stumps are therefore not so obvious as in the *Microsporon* variety. Infections with the several species of endothrix *Trichophyton* are rare in the United States.

The *Trichophyton ectothrix* (animal type of fungus) begins as a surface infection which is followed by involvement of the hair follicles and the hair. It closely resembles infection with the

to produce a spontaneous cure. The subjective symptoms consist of tenderness and pain.

Rarer varieties of ringworm are characterized by baldness (the bald ringworm of Laverne) by small black plugs resembling comedones (black-dot ringworm) commonly due to the *Trichophyton violaceum*, or by scattered spots in which only a few follicles are involved (disseminated ringworm of Acler-Smith).



FIG. 583. Tinea Capitis. (Courtesy of Dr. C. C. Thomas.)

Microsporon lanosum. Occasionally this type of fungus (the *Trichophyton* group usually contracted from cattle and therefore common in rural areas) produces an inflammatory deep-seated carbunclelike tumor (kerion of Celsus). The surface of the tumor ranges from reddish to purple and possesses numerous pinhead to pea-sized pustules. The subcutaneous tissue is honeycombed with gelatinous or mucopurulent material, which may be squeezed through the follicular openings. This type tends to spontaneously disappear in two to three months. Kerion due to *Microsporon* seldom occurs in small children (Kerres and L. act).

In other instances the lesions may be single or multiple. The intensity of reaction to the infection may be sufficient

Diagnosis. All gray scaling patches, containing broken hairs, in the scalps of children should be suspect. Microscopic demonstration of the fungus is essential, however and should be done routinely in every lesion suspected to be due to fungi. Material for examination is removed with a curette in case of a dry and scaly eruption, with a pair of scissors if the nails are involved or with epilation forceps if a hair is to be examined. The material to be examined is placed on a glass slide, and a few drops of a 10 per cent solution of sodium hydroxide are added and a cover slip placed on the slide. The material is macerated by applying a moderate amount of heat obtained from a bunsen burner or flame of a match. The cover slip is then pressed down to remove the excess of sodium hydroxide and to flatten

the material. The slide is then examined under the low power of the microscope. Fungus hyphae and spores are usually plainly seen. If no fungi are found on first examination the specimen should again be examined twelve hours later. Fungi may also be stained or cultured by using



Fig. 586. Herion Cetus. Tricophytic-pronged, putula folliculit of scalp; note a quarter-size rounded, elevated plaque over lower occiput to left of median line. (Courtesy of Dr. Jacques P. Guequierre.)

Sabouraud's proof medium which consists of granulated peptone, crude maltose, agar, and distilled water.

In addition to the microscopic examination of the diseased hair, examination of the scalp under Wood light (black light) in a darkened room is an aid in diagnosis. The Wood light is a valuable asset to the dermatologist for it is used not only in the diagnosis of ringworm but also as a means of determining the extent of the infection, the progress of the disease, and its treatment, and finally as a determinant of cure. Levin and Behr-

man have indicated that Wood light is of especial value in the Microsporon infections. It has been shown that *Trichophyton gypseum* and *purpureum* do not impart fluorescence to the diseased hair; *Trichophyton violaceum*, *sulphureum*, and crateriforme show only a dull white fluorescence which may be overlooked. A negative examination under black light does not, therefore, exclude tinea tonsurans. All scalp disorders in children, when there is scaling and loss of hair, require Wood light examination and, if negative, microscopic and cultural studies are indicated. The method with black light consists in utilization of ultraviolet wavelengths of about 3600 Å which are obtained by passing the beam through a Wood filter composed of glass containing sodium barium silicate and nickel oxide. The source of radiation should give a limited amount of infrared rays, and the filter should prevent the passage of wave lengths within the visible spectrum. The Correx filter of red or blue-purple glass (No. 580 Corning Glass Works) may be substituted for the Wood filter.

In a dark room under this light, the skin fluoresces faintly and is somewhat blue. Margarot and De Vèze were the first to demonstrate the detection of ringworm infection by this method. The fluorescence shows as brilliant luminous pale-green beads on the hairs, contrasting strongly with the dark field; bare scalp areas show as turquoise blue. As lanolin, petrolatum, blood, and some other materials upon the skin also fluoresce, although not confined to individual hairs as in tinea, it is necessary to shampoo the scalp with soap and water followed by alcohol or fat solvent (acetone), to remove these substances. A water-cooled lamp is easier to use as a source of radiation than the air-cooled lamp, but the

latter can be employed by inserting the Wood filter over the small opening in the hood.

By examination under Wood light, it is possible to determine the extent and location of ringworm quickly and accurately where the infection is suspected and where clinical signs have become apparent. Other conditions producing clinical appearances similar to ringworm may thus be promptly excluded, treatment can be instituted and cure attempted by topical application while the infection is still limited to a small area. After treatment, examination under the Wood light may reveal hairs which have escaped, and may thus prevent relapses which otherwise occasionally occur and which are not disclosed until they have produced clinical symptoms. The same technique of value in the diagnosis of *trinea barbae*. The results are usually confirmed by microscopic examination of the fluorescent hairs.

In the microspores, there are well defined circular bald spots, numerous scales, and many hairs broken off at varying levels. In *Tricophyton* or large-pore infections, the bald spots are diffuse irregular more or less of a thinning out of the hair; the scales are scantier and the hairs are broken off at the surface of the scalp and appear as black dots. However infection caused by various fungi often resembles each other. Although clinical inspection may suggest a diagnosis of ringworm and although the causal species may be suspected, cultural studies are often required to make the exact determination. The suspected hairs are placed on a Sabouraud, dextrose, peptone agar slant and after seven days at room temperature, the characteristics of the growth will enable the expert to determine the genus and species (Conant)

Differential Diagnosis. Ringworm of the scalp must at times be differentiated clinically from *favus*, alopecia areata, and seborrheic dermatitis. Only very rarely has ringworm of the scalp been observed in patients over fifteen years of age and, although *favus* can occur in adult life, it is more frequent in childhood. Alopecia areata and seborrheic dermatitis can occur at any age. The distinctive clinical features of ringworm of the scalp are broken-off stumps of hairs, usually in rounded erythematous patches in which there are crusts or pustules and a sparsity of hair. The broken-off hairs are loose and when examined are found to be surrounded by or to contain the ringworm fungus.

In *favus* the principal features are sulfur yellow dry cup-shaped scutula or crusts, associated with atrophic scars and permanent alopecia of varying extent. The hairs in both diseases lose their luster but in *favus* they do not break off near the scalp as regularly as in ringworm. *Favus* in this country generally is seen in immigrants from Poland and Russia. The diagnosis is established by the demonstration of the *Achorion schoenleii*. In alopecia areata the affected patches are completely bald and the skin is smooth and shiny without any signs of inflammation and without scaling. There are no stumps of broken-off hairs, and no fungi are demonstrable. In seborrheic dermatitis the involved areas are covered by fine, dry or greasy scales. When the scales are dry they are branlike and not adherent to the scalp but flake off upon the hair and clothing; whereas, when they are greasy they are adherent to the involved area, which they cover with a thick crust. There is usually no pustulation and there are no broken-off hairs.

Prognosis The prognosis in tinea capitis is favorable as to ultimate cure. In extensive cases, the infection is extremely rebellious to treatment. In cases in which kerion occurs, scarring and baldness may result.

The time required to effect a cure depends on the type of treatment. Epilation is the treatment of choice. When treatment consists of the topical use of drugs the condition may require treatment anywhere from three to eighteen months. Spontaneous recovery usually occurs when adolescence is established. The scalp hair of adults is immune to *Microsporon audouinii* infection but not to *Tricophyton gypsum*, *Microsporon lanosum* or *Achorion schoenleinii*. Rothman and Smiljanic found that with the onset of puberty the sebaceous glands of the scalp start to secrete a sebum which contains, in higher concentration than before, low boiling saturated fatty acids with selective fungistatic and fungicidal action on *Microsporon audouinii*.

Prophylaxis Animal and human sources of infection must be closely scrutinized and eliminated. All infected areas must be treated simultaneously so that dissemination cannot occur. The persons in charge of treatment should observe care not to transfer the affection. Their hands should be washed with soap and water followed by use of an antiseptic lotion both before and after treating the patient's scalp. The patient should have his own comb and brush and these should be rinsed daily in weak ammonia water. Patients should change caps or hats frequently which should have a paper lining so that it may be destroyed daily.

Treatment Some varieties of ringworm of the scalp are curable by thorough and painstaking topical treatment with various antiparasitic remedies, while others clinically similar fail to yield to

the same methods. The response to treatment varies with the nature of the causative organism. Lewis reported a series of studies showing that scalp infections caused by *Microsporon pathogenae* to animals (cats, dogs, horses) are curable by topical measures alone. In his series of cases, all infections with *Microsporon lanosum* and *Microsporon felinum* were cured by epilation with forceps or with local remedies such as iodine crystals, thymol, and oil of cinnamon (each 1 per cent) in petrolatum or 10 per cent ammoniated mercury ointment. Although infections due to *Microsporon audouinii* (human type of fungus) are uniformly unresponsive to topical remedies, they occasionally do respond to chemical agents, and these should be used before drastic depilation with x rays or thallium acetate. When black light reveals but one to several involved areas manual epilation and topical fungicides may be the method of choice.

All cases of scalp ringworm should however be cultured for identification of the organism before treatment is instituted. If it proves to be an organism which is resistant to topical remedies, and if there is extensive involvement the best treatment is epilation with roentgen rays or thallium acetate. If the parasite proves to be one of the animal-type organisms, only topical treatment should be used.

Topical Treatment The following measures are recommended:

1 Clipping hair

2 Daily shampoo with soap and water and removal of all crusts and loose hair

3 Rubbing into the entire scalp, morning and night, a good fungicidal ointment such as 10 per cent ammoniated mercury or 20 per cent sulfur or 1 per cent iodine thymol, and oil of

emmon. The mercury ointment is the least unpleasant and usually is effective.

4. Wearing a skullcap made of material that can be sterilized, throughout the course of treatment. The top of a stocking makes a good cap.

METHOD OF SUTTON AND SUTTON. Sutton and Sutton suggest the removal of all hair in the affected area with epilating forceps. The head should be shampooed daily. After the scalp has been dried, a petrolatum ointment containing 4 per cent salicylic acid and 8 per cent sulfur should be thoroughly rubbed on the affected area. Epilation can be performed daily and should include the hair comprising the margin of the patch.

Waynard suggests the following:

Iodine (crystals)	40
Trichloroethylene	80
Goose grease	300

Schwartz and coworkers claim to have cured 57 per cent of *Microsporon* cases with the following ointment (now commercially available (Parke Davis & Co.))

T 100 parts of molten carbon wax 1800 (Carbide and Carbon Chemical Corp.) is added 5 parts of 11 ounce 100%, 25 per cent (Rohm and Haas Co. Philadelphia, Pa.) and 5 parts of salicylic acid (Blickens Extra, DuPont Co. Wilmington, Del.) The mixture stirred until it becomes a homogeneous mixture, and is then poured into jars and allowed to congeal.

OTHER APPLICATIONS. Of the many topical applications, the following are a few that are useful. They are irritants and should be employed with caution especially to keeping them out of the eyes.

I	
Chrysomelae	
Chloroform q. ad	50
	800
II	
Chrysomelae	
Petrolatum q. ad	50
	800
III	
Tincture of iodine	
Alcohol q. ad	750
	800

IV	
Lozoe crystals	20
Thymol crystals	0.5
Oil of cinnamon	2.5
Liquefied q. ad	800

V	
Crude coal tar	50
Zinc oxide	50
Petrolatum q. ad	800

VI	
Resorcinol	50
Petrolatum (or glycerin) q. ad	800

VII	
Whitfield Ointment	
Salicylic acid	10
Benzoic acid	20
Petrolatum	150
Lanolin	150

VIII	
Salicylic acid	10
Precipitated sulfur	40
Petrolatum q. ad	800

IX	
Betamethol	50
Precipitated sulfur	40
Petrolatum q. ad	800

X	
Salicylic acid	50
Castor oil	10
Alcohol q. ad	800

XI	
Petrolatum	
Tablet salt	150

Thallium Acetate. This drug is still used abroad, and to some extent in this country for its epilating qualities, despite its tendency toward toxic and even fatal complications. It appears to be safest in selected cases—not overly fat children between ages of five and nine years and never to those with organic or renal disease. The child must be accurately weighed and the drug (7 mg per kilogram) dissolved in a little water and given on an empty stomach. The hair is clipped short. The hair loosens about the fourteenth day at which time a cap of adhesive plaster is applied and used

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Treatment Some varieties of ringworm of the scalp are curable by thorough and painstaking topical treatment with various antiparasitic remedies, while others clinically similar fail to yield to

the same methods. The response to treatment varies with the nature of the causative organism. Lewis reported a series of studies showing that scalp infections caused by *Microsporon* pathogenic to animals (cats, dogs, horses) are curable by topical measures alone. In his series of cases all infections with *Microsporon lanosum* and *Microsporon ferrugineum* were cured by epilation with forceps or with local remedies such as iodine crystals, thymol and oil of cinnamon (each 1 per cent) in petrolatum or 10 per cent ammoniated mercury ointment. Although infections due to *Microsporon audouinii* (human type of fungus) are uniformly unresponsive to topical remedies, they occasionally do respond to chemical agents, and these should be used before drastic depilation with x-rays or thallium acetate. When black light reveals but one to several involved areas, manual epilation and topical fungicides may be the method of choice.

All cases of scalp ringworm should, however, be cultured for identification of the organism before treatment is instituted. If it proves to be an organism which is resistant to topical remedies, and if there is extensive involvement, the best treatment is epilation with roentgen rays or thallium acetate. If the parasite proves to be one of the animal-type organisms, only topical treatment should be used.

Topical Treatment The following measures are recommended:

1 *Clipping hair*

2 *Daily shampoo* with soap and water and removal of all crusts and loose hair

3 *Rubbing* into the entire scalp, morning and night, a good *fungicidal ointment* such as 10 per cent ammoniated mercury or 20 per cent sulfur or 1 per cent iodine, thymol, and oil of

after 3 of an epulating dose, but there have been no cases of permanent alopecia in our experience. It probably requires 800 (13½ E.D.) or more to cause permanent alopecia. A mild erythema appearing each after treatment and lasting to or three weeks has resulted in the reappearance of the hair. About more than eight days. X-ray treatment is sometimes erroneously held responsible for scarring and permanent destruction of the hair follicles in small areas caused by the disease itself. To avoid any injustice or criticism on this point, it is recommended that the parents of all patients with suppurative deep lesions should be told of the possibility of slight scarring before any treatment is given.

X-ray treatment causes the hair to fall out but has no direct effect upon the fungus, except that when the hairs fall the fungi which inhabit them are immediately removed. Immediately after epilation, antiseptic measures are instituted to destroy the few spores which remain in the hair follicles and on the scalp, with the purpose of avoiding relapses and reinfection. Remedies are more effective if all crusts are removed prior to their application by the use of pumice or starch paste composed of 4 table-spoonfuls of starch mixed with little cold water and then stirred continuously into 1 pint of boiling water is added, so that jelly is formed. To this, 1 teaspoonful of boracic acid and 1 table-spoonful of glycerine are added. A dressing of this paste is applied to the crusted scalp for four to six hours, and then it is washed off. Of antiseptic remedies, 5 per cent sulfur ointment and 5 per cent ammoniated mercury ointment are popular the latter being preferable. The strength of these ointments is gradually increased until all signs of infection have disappeared. It is often necessary to increase the strength of the sulfur ointment to 8 per cent, especially on incomplete epilated scalp, and even the addition of tar or salicylic acid is sometimes of value. In infected cases wet dressings of solutions of aluminum acetate or ammoniated mercury ointment are indicated, and small fluctuating abscesses must occasionally be incised and drained. During the entire treatment the scalp should be shampooed every morning and the local medication should be applied afterward, as well as at bedtime.

Regrowth of hair noticeable from two to four months after epilation. This interval is of importance in allowing differentiation of the follicles to take place before the new hair forms. Tschernakow has stated that there must be depression of the activity of the papilla following the epila-

tion due to otherwise the new growth of hair would appear at the surface in thirty days. The regrowth may appear earliest and thickest in the areas which are diseased and may vary from the original hair as to texture shade of color or curliness.

A cure is assured if the epilation is complete and the scalp is rid of clinical evidences of the disease by means of antiseptic remedies before the regrowth of hair becomes visible. However many patients do not follow their requirement are also cured. Of this group the most unfavorable are those in whom the hair regrows before the scalp is free of lesions. Before being discharged, all patients who have any questionable areas should have Wood light and microscope examination, preferably done 1 week after the stopping of local remedies.

The failures to obtain complete epilation are usually due to excessively short scissor-like clippers, inefficient clipping of the hair before the exposure, thus starting out some of the radiation or insufficient focusing, with resultant errors in the overlapping. Whenever residual tufts of hair persist after epilation, particular care must be exercised for they are common causes of failures. Sometimes small fringes of hair persist on the back of the neck, about the ears, at the anterior margin of the scalp or vertex, they render almost always an evidence of imperfect technique or restlessness of the child. It is not safe procedure to give (or their parents) such tufts; even the slightest ones must be epilated by forceps. Failures to cure may also result from reinfections from other children, old hats, or pets, especially cats, dogs, and canaries.

In case of failure to epilate the scalp, repetition of roentgen-ray treatment cannot safely be made for at least four months, when an epulating dose may be given again, with special precaution to avoid the mistakes which led to difficulty in the first attempt. When epilation has been more or less thorough, but the disease is not cured due to neglect of such precautions as manual epilation of few persistent hairs or the thorough use of antiseptics, or due to reinfection, six months should elapse before the first and second x-ray treatments.

Tinea Imbricata

SYNOGYMES *Barrois ringworm*,
Tekalon ringworm.

Tinea imbricata is a dermatomycosis caused by fungi of the genus *Endodermophyton*.

to remove the loosened hair. When all hair is out the scalp should be cleaned daily with acetone soap and water and a fungicidal ointment used to prevent reinfection. Retreatment may be necessary but never before four to six weeks have elapsed.

Ray Epilation. A highly successful method has been suggested by George Mackee. It is applicable to all children between the ages of two to eleven years.

Directly preceding radiation, the entire hair is combed flat by the comb above the skin surface. A line is necessary. The scalp should then be washed with green soap until the crusts are removed. It may be marked for epilation with skin pencil and either a tape measure or a marker to indicate the Kienbock-Adamson point as a guide.

It is convenient to treat the vertex point first. For this purpose the child lies prone with the chin supported so that the vertex point is uppermost. The eyes are banded with lead spectacles and the shoulders with lead-rubber. Point C at the base of the occiput is not treated. To obtain a proper position for the child, pillow is inserted under the chest and the head is strongly flexed forward so that it hangs down over the edge of the pillow giving good exposure to the point at the base of the occiput. Shield the same as in the previous position. For the other parts, the child lies on his back. The lateral points D and E or the ears, are conveniently treated next. The operator begins with the right side the right ear being held forward with adhesive strapping so that it is withdrawn from the field and lies flat on the cheek. The child's head is turned to the left, the face abducted and focus is made. After exposure the head is turned to the right and the left ear is held forward with adhesive. The holding is similar to that of the preceding exposure. The last point, H, is treated is most difficult because child objects to his face being covered with heavy heat of metal. The median frontal point is prominently exposed by raising the head on two or three pillows, so that the chin is resting on the chest while the child is lying on his back. The eyes are covered with lead spectacles and the face with lead rubber sheeting.

Causes of anxiety or fear on the part of the child are often of trivial origin. Any mention of pain or fear gives a sense of terror and therefore

such expressions should be carefully avoided. If pillows are placed against the child's nose breathing is often obstructed, which causes great restlessness and anxiety.

On angular heads special care should be exercised to arrange the focusing and overlapping so that equal distribution is given to the dose. It is very important that the direct beam of the radiation be at right angles to the sagittal or frontal planes and that the direct rays of the vertex exposure be in line with an imaginary perpendicular erected through the vertex point from the center of the area included within the circumferential line.

Dosages slightly smaller than those necessary to produce an erythema of the scalp will cause temporary epilation. Our epilating dose is 330 r unfiltered, with mechanical rectification, with Coolidge universal type broad focus tube operating at 100 k. 3 mm., at 8-inch anode-skin distance. With Kienbock rectification the ray quality is softer and the epilation dose according to Lane and MacKee is about 300 r. Variations in the amount of overlapping due to differences in the size of heads are controlled by changes in the anode-skin distance. Heads with a circumference of 10 inches or over are treated with an 8-inch skin-target distance. For heads with a circumference of 11 inches, it is advisable to use a skin-target distance of $6\frac{1}{2}$ inches in order to avoid too much overlapping. When the head is smaller than this, the dose should be slightly reduced. Adult heads with a circumference of 22 or more inches require a slightly larger dose. It is sometimes advantageous in adults to use a skin-target distance greater than 8 inches or even seven instead of five areas.

After proper dosage the hairs begin to loosen and fall in about seventeen days, carrying with themumberless spores. In order to prevent seedling spreading of the disease at the time of defoliation patients under treatment are instructed to wear bit head coverings, which are easily made from paper bags or the tops of white stockings. These are changed daily the used ones being burned. About the seventeenth day when the hair is loose the scalp is covered with adhesive plaster in form of skull cap. This is left in place over night and the following day is pulled off and burned, the loose hairs being pulled out with the adhesive cap. If the epilation has been completely successful, the scalp is then hairless. However, complete defoliation is not imperative for a cure in the majority of cases.

There is considerable margin of safety between the epilating dose and that which causes permanent alopecia. Occasionally the hair will fall out

ual patches are yellowish or brownish maculae, which gradually increase in size and coalesce to form extensive scaling lesion. At times the surface is smooth, particularly in persons who bathe once or twice a day. In other instances, a follicular tendency is a marked feature of

atrophy subsequently appears. This condition, *achromia parasitica* (Pardo-Castello)—an epiphenomenon in tropical climates in the evolution of other epidermycoses as well—is characterized by the appearance of a variable number of gray white variously sized, rounded or



Fig. 537 *Tinea Versicolor*. (Courtesy of Dr. Jacques P. Guequiere.)

the eruption. Mild itching may be present, however inflammation is absent. In brunettes the affected patches may be lighter in color than the rest of the integument.

Achromia Parasitica. When patients with *tinea versicolor* expose themselves to sunlight for periods sufficient to tan the areas of the skin not affected by *tinea versicolor* an apparent pigmentary

oval spots, especially on the back, neck, and face. The fungi can be found in microscopic examination of the fine scales usually present on the achromic areas. The condition is probably due to the prevention by the fungus masses of the normal processes of pigmentation under exposure to sunlight.

There is also a nonparasitic *achromia* occurring in the form of dirty whitish

Incidence It occurs in moist tropical climates

Etiology The *Endodermophyton tropicale* is the usual cause

Symptoms This disease is characterized by scalp patches which cover large parts of the covered surface of the body and which assume a circinate or concentric arrangement. This disease begins with one or more reddish macules which quickly become papular. The affected areas enlarge and in a few weeks they are a centimeter or more in diameter. In the center of the area the epidermis breaks and gives rise to rough patches, the margins of which are formed by upcurled lamellae. The free edges of the lamellae incline toward the center of the exfoliating patch. Repetitions of this process result in the development of concentric rings which coalesce to form a mottled and imbricated appearance. Chronic cases present brownish concentric rings with large scales. The disease resembles ichthyosis. The face, scalp, palms, soles and nails are not involved. Subjective symptoms consist of intense itching.

Diagnosis The disease is differentiated from *ichthyosis* and other varieties of tinea infection. Examination of the scales and the clinical picture are diagnostic.

Prognosis The prognosis is guardedly favorable because the disease is difficult to treat successfully.

Treatment Treatment consists of the use of *Castellani's fuchsin paint*, *chrysarobin* or *Deek's ointment*.

Castellani's Paint

Boric acid	1.0
Acetone	5.0
Resorcinol	10.0
Sol. carbolfuchsin (Ziehl-Neelsen)	100.0
q.s. ad	
M	

Deek's Ointment

Salicylic acid (fine powder)	1.0
Bismuth subnitrate	10.0
Mercuric salicylate	1.0
Oil eucalyptus	2.0
Petrolatum	40.0
Wool fat	5.0
M	

Tinea Versicolor

SYNONYMS: *Pityriasis versicolor*, *chromophytosis*, *dermatomycosis furfuracea*.

Tinea versicolor is a vegetable dermatomycosis characterized by yellowish or brownish patches, usually occurring on the chest and shoulders.

Incidence The disease occurs in adults of both sexes. It is seen more frequently in people who perspire freely; hence it often occurs in tuberculous patients.

Etiology It is caused by the *Microsporon furfur* (*Malassezia furfur*) which is a vegetable fungus.

Pathology The fungus is easily seen in scrapings which have been immersed in a solution of 10 per cent potassium hydroxide. The lesion consists of almost pure culture of the fungus and scales. Microscopically strands of short angulated hyphae and large numbers of variously sized spores are seen. Their aggregation in clusters is characteristic. The organism may be cultured in a keratin-sweat medium (Cornbleet). The fungus is situated in the upper and middle layers of the epidermis, in the follicles and sweat orifices. The hair is never affected.

Symptoms Fawn-colored finely desquamative, guttate, nummular or palm-sized patches are characteristic of this affection. These tend to run together and usually occur on the upper part of the trunk. Although the eruption may spread over the shoulders, back, chest, axillae, and upper abdomen, rarely are the face and scalp affected. The individ-

of *parasitoides*. The application of a 5 per cent aqueous solution of *sodium hypomphite* followed immediately by a 5 per cent aqueous solution of *tartaric acid* is an excellent method of treatment.

Other satisfactory method of treatment include the application of a 3 per cent *tincture of iodine* rubbing the affected parts with *resorcin* twice daily

followed immediately by the application of 25 per cent aqueous solution of *sodium hypomphite*. The application of an ointment consisting of 3 per cent *precipitated sulfur* and 2 per cent *salicylic acid* is also a favorable prescription.

Ultraviolet baths with first-degree erythema dosage are also an effective means of producing a complete cure.

TROPHEDEMA (MILROY-MEIGE)

SYNONYMS *Chronic hereditary edema, lymphedema, or trophedema, neuroarthritic pseudolephanthiasis.*

Etiology This condition is familial hereditary or congenital, but is some-

times acquired. The exact cause is unknown.

Symptoms Trophedema is usually symmetrical, and generally involves the lower limbs. It may be limited to the ankles or dorsa of the feet. Such abortive forms of the disease are commoner than is generally believed. The affected parts, shortly after birth or later become large as a result of persistent edema which is painless and solid and which may pit slightly on pressure. Once established, the edema is persistent and apt to be progressive. The acquired form commonly develops after puberty in young girls.

Diagnosis This is usually not difficult. Trophedema differs from *elephantiasis* by the fact that the skin shows no lymphatic varices, is always smooth, and has normal color. There is no history of recurrent inflammatory crises.

Treatment Except for massage and compression with rubberized stockings, treatment is of little avail.



Fig. 898 Chronic Hereditary Trophedema (Milroy Disease) Unusually marked example; mild forms of this disease are not rare.

A number of cutaneous lesions differing widely in their appearance and course of development are directly or indirectly the result of infection by the Koch bacillus (*Mycobacterium tuberculosis*)

TUBERCULOSIS CUTIS

Varieties The more common forms of skin tuberculosis are lupus vulgaris, acrofuloderma, tuberculous ulcerosa, tuberculous verrucosa, tuberculous vegetans et frambiformis, and milium tuberculosis.

spots, this develops on tanned skins which have not been exposed to sunlight for several months. This leukoderma factitia appears to be a normal phenomenon in some persons as detanning or depigmentation progresses in the fall and winter. It represents an irregular rather than a uniform depigmentation of the



Fig. 588: *Achromia Parasitica* (in *tinea versicolor*) (Courtesy of D. Jacques P. Cnequiere)

tanned area. The condition must not be confused with *achromia parasitica* nor with *vittigo*.

Diagnosis. *Tinea versicolor* must be differentiated from *seborrheic dermatitis*, *sypilis*, *chloasma*, *lentigo* and *vittigo*. In *seborrheic dermatitis* the patches have an erythematous, yellowish tint and the scales are greasy. *Macular sypilis* is characterized by rose-red or pink lesions, irregularly round or oval, occurring principally on the sides of the trunk and flexor surfaces. General adenopathy, mucous patches, and a positive Wassermann reaction are usually present in

sypilis, and never occur in *tinea versicolor*. *Chloasma* usually occurs on the face, and scaling and itching are always absent. *Lentigo* is likewise free of scales and itching. *Vittigo* is usually present on the forehead, face, and dorsum of the hands. Scaling and itching are also absent. In case of doubt microscopic examination of the scales always establishes the true diagnosis.

Prognosis. The disease responds readily to treatment. If untreated, it continues for many years, although in the aged a spontaneous cure occurs. It is less noticeable in winter than in summer. Relapses are common.



Fig. 589: *Achromia Parasitica*, (Courtesy of Dr. V. Pardo Castello.)

Prophylaxis. Prophylaxis consists in the avoidance of contact with those already affected. Clothing worn by affected persons should be discarded and never used by others.

Treatment. The affected areas should be frequently and thoroughly scrubbed with soap and water followed by the use

decades in exceptional cases. It is commoner among females and very rarely occurs in more than one member of the same family.

Climate appears to play an important role in the incidence of lupus vulgaris because of its common occurrence in

The cellular elements of a lupus nodule undergo coagulation necrosis and fatty degeneration in consequence of the avascular nature of lesions. This is followed by scar tissue formation.

Symptoms The primary focus of infection is a macule or papule, although the occurrence of multiple lesions is not uncommon. Lesions may occur on the face after an acute specific fever simulating that of measles. The appearance of facial lesions is referred to as *lupus dissemminatus*. Lesions spread by peripheral extension and adjacent lesions coalesce to form large plaques. Cystic and festooned lesions result from central cicatrization of nodules and their peripheral extension.

Subjective symptoms are negligible. The increase in size of lesions is gradual hence little change occurs over a long period of time.

The primary lesion is the outstanding pathognomonic objective symptom. It is seen best in daylight under the pressure



Fig. 591. Lupus vulgaris. Healed with scars and typical break-down nose. (Courtesy of Dr. Howard Fox.)

cold climates and rarely in the tropics.

Inadequate diet, insufficient food, bad hygiene and want of sunlight are important factors in diminishing the individual's resistance to infection. The most destructive forms of this disease occur among children living in undesirable environment favoring infection.

Pathology A nodule of lupus vulgaris consists of miliary tubercles identical to those of phthisis pulmonalis. This histopathology presents epithelioid cells, plasma cells, and giant cells surrounded by round cell infiltration.



Fig. 592. Lupus vulgaris. Twelve years duration.

of a glass slide or disk (diascopic examination) because this pressure diminishes the accompanying hyperemia, thus bringing out the nonvascular nature and apple-jelly (yellowish brown) color of lesions.

Etiology and Pathogenesis The *Mycobacterium tuberculosis* invades the skin in one of the following ways

1 By direct entry of the bacillus through skin abrasion. This occurs in the majority of cases of lupus vulgaris and tuberculous verrucosa.

2 By infection from broken-down tuberculous glands and from sinuses leading to tuberculous foci in bones and joints. This occurs in acrofuloderma.

3 By extension from mucous membranes. This occurs in connection with the nasal cavity in certain cases of lupus vulgaris.

4 By autoinoculation from open tuberculous lesions in the lung bowel or genitourinary tract. This occurs in tuberculo ulcers.

5 Through the blood stream. This is the method by which miliary tuberculosis of the skin takes place. The blood stream may also be the avenue by which lupus vulgaris is produced.

Specific antibodies in the normal skin make it a poor medium for tuberculous infection. Skin tuberculosis is an accident. The type of skin tuberculosis is determined by virulence of the bacillus and its cultural terrain. Cutaneous tuberculosis is a rarity in sanatoria for pulmonary tuberculosis.

Diagnosis The bacillus tuberculosis is usually present in the tissue in all cutaneous lesions of tuberculosis. It or the results of its presence can readily be demonstrated by one of the following laboratory methods.

1 By microscopic examination of properly stained sections.

2 By a positive reaction in a guinea pig inoculated with infected tissue.

3 By the local reaction resulting from the Mantoux test or intradermal injection of old tuberculin 0.1 cc of 1:1000 dilution or the intradermal injection of

the purified protein derivative of tuberculin (0.1 cc).

4 By the von Pirquet test of applying a 25 per cent solution of old tuberculin in isotonic saline solution to the scarified skin or the Vollmer patch test (Lederle).

5 By the histopathology of the tissue and clinical features.

Lupus Vulgaris

SYNONYMS: Tuberculous cutsis in *poa*, lupus form lupus infectiois granulomata.

Lupus vulgaris is the most common form of cutaneous tuberculosis. It is a tuberculous granuloma of the skin and adjacent mucous membrane. The primary lesion is a small, soft nodule varying in color from yellowish red to reddish brown. Lesions may be macular or moderately elevated. They may or may not be scaly. Various sized plaques and patches are formed by multiplication and coalescence of nodules. Tissue destruction occurs from ulceration and subepidermal cicatrization.

Etiology Lupus vulgaris is the result of infection by the *Mycobacterium tuberculosis hominis*, although the *Mycobacterium tuberculosis bovis* has been the causative agent in exceptional cases. The disease is produced rarely by avian *Mycobacterium tuberculosis*. Lupus vulgaris appears in the majority of cases as the result of accidental inoculation following trauma as in abrasion, tattooing, circumcision etc.

Darier is of the opinion that lupus vulgaris is due to infection of allergic individuals. The presence of this susceptibility would explain why phthisis pulmonalis does not always follow lupus vulgaris.

The disease occurs in the first decade of life although it has appeared in later

decades in exceptional cases. It is commoner among females and very rarely occurs in more than one member of the same family.

Climate appears to play an important role in the incidence of lupus vulgaris because of its common occurrence in



Fig. 891. Lupus Vulgaris. Healed lesions and typical break-like nose. (Courtesy of Dr. Howard Fox.)

cold climates and rarely in the tropics. Inadequate diet, insufficient food, bad hygiene and want of sunlight are important factors in diminishing the individual resistance to infection. The most destructive form of this disease occurs among children living in undesirable environments favoring infection.

Pathology. A nodule of lupus vulgaris consists of miliary tubercles identical to those of phthisis pulmonalis. This histopathology presents epithelioid cells, plasma cells, and giant cells surrounded by round cell infiltration.

The cellular elements of a lupus nodule undergo coagulation necrosis and fatty degeneration in consequence of the avascular nature of lesions. This is followed by scar-tissue formation.

Symptoms. The primary focus of infection is a macule or papule, although the occurrence of multiple lesions is not uncommon. Lesions may occur on the face after an acute specific fever simulating that of measles. The appearance of facial lesions is referred to as *lupus disseminatus*. Lesions spread by peripheral extension and adjacent lesions coalesce to form large plaques. Crater and festooned lesions result from central cicatrization of nodules and their peripheral extension.

Subjective symptoms are negligible. The increase in size of lesions is gradual, hence little change occurs over a long period of time.

The primary lesion is the outstanding pathognomonic objective symptom. It is seen best in daylight under the pressure



Fig. 892. Lupus Vulgaris. Twelve years duration.

of a glass slide or disk (diascopic examination) because this pressure diminishes the accompanying hyperemia, thus bringing out the avascular nature and apple-jelly (yellowish brown) color of lesions.

Seventy-eight per cent of cases of lupus vulgaris occur on the face, nose, cheeks, and auricles. The neck is next in incidence. The hairy scalp, forehead and upper eyelids are rarely invaded.



Fig. 593: Lupus vulgaris Erythema toides (Vidal and Leloir). Lupomata are readily seen under vitropressure.

About 8 per cent of the cases of lupus vulgaris presenting for treatment in the London Hospital showed involvement of the extremities and trunk. The disease is exceedingly rare on the palms, soles, axillae, genitals and about the anus. About 43 per cent of patients attending the London Hospital Clinics showed mucous membrane involvement. Mucous membrane involvement occurred in 80 per cent of cases of lupus vulgaris presenting for treatment in the Finsen Institute of Copenhagen.

A present or past history of pulmonary tuberculosis is usually present in 63 per cent of patients suffering from

lupus vulgaris. A history of tuberculosis in the forebears occurs in the remaining 37 per cent.

Lupus planus is a descriptive term identifying flat patches. Elevated plaques are known as *lupus discoides*. An erythematoid variety of *lupus planus* may require histologic study to differentiate it from *lupus erythematosus*. Dry patches of lupus vulgaris are called *lupus nonexedens*. *Lupus ulcerosa* (*exedens*) is a form of lupus vulgaris in which nodules and patches undergo ulceration.

Extensive edema, hypertrophy, hypoplasia, telangiectasis, lymphangitis, and lymphadenitis identify various forms of lupus vulgaris known respectively as *lupus edematosus*, *lupus hypertrophicus*,



Fig. 594: Lupus vulgaris in child. On ven duration. Note modules which are ppie jelly or yellowish-brown in color under glass pressure.

lupus papillomatosus, *lupus elephantiacum*, *lupus tumidus*, and *angiolupod*.

Lupus exfoliativa (*lupus psoriasisformis* or *tuberculeux* of Vidal and Leloir) is characterized by patches which are

scaly depressed in the center from cicatrix formation, and spread by peripheral extension.

Lupus sclerosus (*lupus fibrosus*) presents fibrous metamorphosis leading to sclerosis with contraction of tissue and terminates in hideous deformities.



Fig. 895 *Lupus Vulgaris* (archiform, squinate, nodular) lesions of upper lip, on right ala nasi, and in left nostril, of four years duration. Wassermann and Kahn, blood-serum reactions were negative.

Lupus exedens manifests oval or circular ulcers discharging pus. The crusts of these lesions assume various thick nesses and present yellowish-gray discoloration.

Lupus serpyginosus *lupus superficialis* *lupus profundus* *lupus gangrenosus* *lupus mutilans* *et phagedenicus* are descriptive terms identifying various forms of *lupus exedens*. The virulence of the offending organism and its cultural terrain determine these forms of *lupus exedens*. The lesions occurring on the body are more extensive but less destructive



Fig. 896 *Lupus Vulgaris*. Extensive, with involvement of labial and gingival mucosa and conjunctiva of left eye.

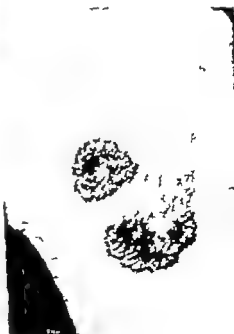


Fig. 897 *Lupus Vulgaris*. Of buttocks, stimulating secondary syphilide.



Fig. 598: *Left:* Lupus Vulgaris. In a child of one year's duration. Not nodules which are yellowish brown in color. *Right:* Lupus Vulgaris Exedens.



Fig. 599: Lupus Vulgaris. *Left:* Small patch in region of left (outer angle) eyebrow and temple. *Right:* The lupomata almost stand out in this type.

than those occurring on the extremities and face. The bones and muscles are not involved in lupus vulgaris. The cartilage may be involved and undergo destruction in lupus vulgaris.

Lupus vulgaris ends in white thin smooth permanent scars. Ulceration and keloid formation may however occur

Lupus of the Mucous Membranes
The nasal mucous membrane is a frequent site of lupus vulgaris. Nasal lupus may spread to the lips and involve the buccal mucosa, the gum tissue, palate, larynx and tongue. Mucous membrane lesions appear as moist papillary growths or as granulating patches which undergo

ulceration and occasional excoriation. The usual site of predilection in the lip region is the mouth commissure in which fissuring is an outstanding feature. Lesions of the conjunctivae are a continuation of the facial lesions and may lead to ectropion or entropion and may end in corneal involvement and destruction of the eye.

Lesions consisting of military ulcers and fissuring occur about the anus.

The progress of mucous membrane lesions is slow showing no tendency to heal and becoming very painful from secondary infection.

Course of Lupus Vulgaris. Lupus vulgaris is a slowly progressing disease which may advance rapidly from mixed infection. Cases of thirty or forty years duration are not uncommon. Recurrences are quite common. Grave deformities follow destruction of the nasal cartilage. Limitation of joint movement, mutilation of the phalanges, etc., occur in lupus exedens.

Complications: Erysipelas is not an uncommon complication of lupus vulgaris. Benefits have been reported from a complicating erysipelas. Erysipelas may complicate scrofuloderma, tuberculous ulceration, and verrucous tuberculous of the skin. Lymphangitis or lymphadenitis may be present in any type of lupus vulgaris. Pulmonary complications are frequent sequelae of nasopharyngeal lupus. Visceral and joint tuberculosis are infrequent sequelae. Carcinoma occurs in from 2 to 5 per cent of cases of lupus vulgaris.

Diagnosis. Lupus vulgaris is not to be confused with lupus erythematosus, tertiary syphilis, prosy blastomycosis, leishmaniasis, porotrichosis, lupoid syphilis, basal-cell epithelioma. The diagnostic feature of lupus vulgaris is the primary lesion characterized under micro-

pressure by the so-called apple-jelly like nodule which differentiates it from other cutaneous disturbances. No difficulty should arise in diagnosing the disease because it advances slowly, starts usually in early life and takes years to produce tissue destruction.

Scrofuloderma

SYNONYMS: Tuberculous cutis, colligativa, tuberculous gumma, necrotic gumma, lupus cutis.

Etiology. The term "scrofuloderma" identifies cutaneous lesions arising in connection with local action of the *Mycobacterium tuberculosis* infecting

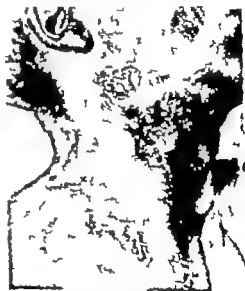


Fig. 600 Scrofuloderma.

the skin from tuberculous foci within the body. Scrofuloderma occasionally arises via the blood stream, especially in cases of military tuberculosis.

Symptoms. Scrofuloderma occurs anywhere in the body. The sites of predilection are the neck, groins, joints, and the cutaneous surface over the bones which are frequent sites of tuberculosis.

Scrofulodermata begin as painless nodules within the subcutaneous tissue.

The commonest form of scrofuloderma is met in the neck where the cervical lymph nodes have been tuberculous for a long period. These nodes become closely matted and adhere to the overlying skin. The skin assumes a purplish red hue and breaks down with the formation of fistulas, sinuses, and ulcers.

The ulcers are usually linear in outline although they may become circular. They are covered with pale flabby granulation tissue. The borders are blue undermined, tumid, and occasionally inverted. The base of an ulcer is soft and firmly attached to the underlying tissue. Induration is absent. The discharge is fetid, seropurulent, and contains the *Mycobacterium tuberculosis*.

Isolated subcutaneous indurations of the skin resembling scrofuloderma are designated "scrofulous gummas." Lesions of this description may occur independently of any subjacent tuberculous process or precede the development of scrofuloderma. They occur in children or in those of low vitality. The most frequent sites of scrofulous gummas are the extremities where they develop slowly and finally break down to form indolent ulcers with undermined edges and necrotic bases.

Tuberculosis Ulcerosa

SYNONYMS: *Tuberculosis cutis*
vera, *tuberculosis cutis orificialis*.

Etiology. A tuberculous ulcer is a clinical curiosity because of its rarity. It is usually associated with pulmonary and intestinal tuberculosis or with tuberculosis of the kidney and bladder. The sites of predilection are the mucocutaneous junctions of the lips, nose, anus, and genitalia. Tuberculous ulcers occur also on the mucous membrane of the

mouth, gingiva, tongue, and pharynx, as well as on the glabrous skin.

Smears and cultures from tuberculous ulcerosa reveal the *Mycobacterium tu-*



Fig. 601: Tuberculosis of the Tongue



Fig. 602: Tuberculous Ulcer

berculosis. This organism can also be obtained from affected lymph nodes as well as from primary lesions.

Symptoms. Tuberculous ulcerosa first appears as one or more small, dull red, dermal granulomas which break down



Fig. 603 Atypical Tuberculous Ulcer. Of fifteen years' duration; guinea-pig inoculation with portion of the tissue removed from edge of ulcer produced tuberculosis.

and form shallow ulcers with delicate undermined borders. The ulcer is oval and serpiginous, and may attain a diameter of many centimeters. The base is irregular and studded with minute yellow granules. The ulcer is usually painless, although it is extremely tender on pressure. Pain is experienced on movement of the involved parts and wherever juxtaposition of the lips produces friction. Ulcers remain shallow and show no tendency to spontaneous healing. The regional lymph nodes enlarge early and become quite prominent. The following

forms of tuberculous ulcerosa are recognized.

1. *Fissured tuberculous ulcerosa* appears at sites of inoculation. This may occur as a painful fissure about the median aspect of the lip, the side of the tongue, or the anus.

2. *Vegetative tuberculous ulcerosa* is met with in conjunction with dry papillomatous growths of the tongue and anus.

3. The so-called *tuberculous chancre* or *primary tuberculous complex* is rare. It is seen among very young people who have no tuberculosis elsewhere in the body. Most of the reported cases have



Fig. 604 Primary Inoculation Tuberculosis of Lip (primary tuberculous complex). Note follicles over right submaxillary region due to lymphadenopathy. Koch bacillus found; dark-field negative.

followed inoculation from infected saliva of carriers (kissing, circumcision customs, etc.) It has been reported to occur on the nose, cheek, lip, chin, brow and genitalia. The lesion resembles an acneiform pustule. It may also assume the appearance of a sharply circumscribed raised nodule in which central necrosis occurs,

following which the appearance is that of a crusted ulcer with a somewhat elevated rolled in border. The tendency is for this lesion to undergo spontaneous regression. Regional adenitis is quite common however. Suppuration with involvement of the overlying skin is frequent.

Diagnosis: Tuberculous ulcers are differentiated from simple ulcers, lumban chancre, mucous patches, cutaneous diphtheria and epithelioma by demonstrating the tubercle bacillus in smears and in the biopsy of ulcers also by the blood serology and the finding in the lesions of the *Spirochaeta pallida* or the diphtheria bacillus.

Tuberculosis Verrucosa

SYNONYMS: *Lept verrucosus*, *an tomical tube de verrues necro-génice*.

Tuberculosis verrucosa is characterized by warty lesions arising from direct inoculation by the *Mycobacterium tuberculosis hominis* or *bovis*. It may occur in cases of phthisis pulmonalis following autoinoculation.

Incidence: The victims of tuberculosis verrucosa are doctors, nurses, pathologists, veterinarians, divers, tanners and butchers.

Etiology: This form of cutaneous tuberculosis is more commonly seen in individuals who come in contact with tuberculous patients, their exudates, and contaminated belongings.

Pathology: The histopathology of tuberculosis verrucosa consists of the characteristic microscopic appearance of tuberculous tissue, showing giant cell and the *Mycobacterium tuberculosis*. Guinea pig inoculation is positive for tuberculosis.

Symptoms: The sites of predilection for tuberculosis verrucosa are the dorsum of the hands, fingers, wrists, thumbs,

forearms and limbs. The perianal and peroneal regions are involved less frequently.

The lesion first appears at site of abrasion as a horny nodule, a papule, or a vesicopustule. It undergoes rapid infil-



Fig. 605: *Lupus Verrucosus*. Of fifteen years duration.

tration and wart formation. The base is deeply scathed and encircled by an inflammatory red violaceous areola.

Hyperkeratoses and the appearance of vegetations are probably due to secondary infection by pyogenic cocci of low virulence. The lesions may remain single or may spread to form large palm-sized plaques. Miners suffering from phthisis pulmonalis show occasional multiple lesions of tuberculosis verrucosa distributed over exposed surface of the body. Cases have also been reported in which infection followed laundering handkerchiefs used by tuberculous patients.

The appearance of these lesions suggests an infected wound.



Fig. 606 Tuberculosis Verrucosa Cutis.

The regional lymph nodes enlarge early in the course of the disease.

This variety of cutaneous tuberculosis is most frequently due to external contamination and occurs as a single lesion if it arises via the blood stream, the lesions are usually symmetrically located on extensor surfaces of the extremities, usually in the region of joints.

Diagnosis: Tuberculosis verrucosa is confused with blastomycosis, chronic forms of pyoderma, late syphilis, hypertrophic lichen planus, nododerma, and bromoderma. The demonstration of the bacillus of tubercle in the tissue and in exudates establishes the diagnosis.

Tuberculosis Vegetans et Framboesiformis

Tuberculosis vegetans and framboesiformis follows direct infection by the



Fig. 607 Framboesiformis Tuberculosis Cutis. Note lesion on scalp.

following which the appearance is that of a crusted ulcer with a somewhat elevated rolled in border. The tendency is for this lesion to undergo spontaneous regression. Regional adenitis is quite common however. Suppuration with involvement of the overlying skin is frequent.

Diagnosis. Tuberculous ulcers are differentiated from simple ulcers, lumpy chancre, mucous patches, cutaneous diphtheria, and epithelioma by demonstrating the tubercle bacillus in smears and in the biopsy of ulcers also by the blood serology and the finding in the lesions of the *Spirochaeta pallida* or the diphtheria bacillus.

Tuberculosis Verrucosa

SYNONYMS: *Lupus verrucosus*, anatomical tubercle verrucosa necrogenica.

Tuberculosis verrucosa is characterized by wart-like lesions arising from direct inoculation by the *Mycobacterium tuberculosis* hominis or bovis. It may occur in cases of phthisis pulmonalis following autoinoculation.

Incidence. The victims of tuberculosis verrucosa are doctors, nurses, pathologists, veterinarians, divers, tanners, and butchers.

Etiology. This form of cutaneous tuberculosis is more commonly seen in individuals who come in contact with tuberculous patients, their exudates, and contaminated belongings.

Pathology. The histopathology of tuberculosis verrucosa consists of the characteristic microscopic appearance of tuberculous tissue showing giant cell and the *Mycobacterium tuberculosis*. Guinea pig inoculation is positive for tuberculosis.

Symptoms. The sites of predilection for tuberculosis verrucosa are the dorsum of the hands, fingers, wrists, thumbs,

forearms and limbs. The perianal and perioral regions are involved less frequently.

The lesion first appears at site of abrasion as a horny nodule, a papule, or a vesicopustule. It undergoes rapid infil-



Fig. 605. *Lupus Verrucosa*. Of fifteen years duration.

tration and wart formation. The base is deeply seated and encircled by an inflammatory red, violaceous areola.

Hyperkeratoses and the appearance of vegetations are probably due to secondary infection by progenic cocci of low virulence. The lesions may remain single or may spread to form large palm-sized plaques. Miners suffering from phthisis pulmonalis show occasional multiple lesions of tuberculosis verrucosa distributed over exposed surface of the body. Cases have also been reported in which infection followed landrering bandkerchiefs used by tuberculous patients.

The appearance of these lesions suggests an infected wound.

losis. Bone visceral, or glandular tuberculosis is often present. The disease is the common sequel of scarlet fever and measles.

a few millimeters to 1.5 cm. in diameter. The papules are usually located around a hair follicle, are flat topped, and occasionally covered with a scale. They are



Fig. 611 Lichen Scrofulaceorum.

This variety of skin tuberculosis is characterized by papular groups distributed on the front, back, and sides of the trunk, rarely on the extremities. The individual papule is small, varying from

reddish brown. They may appear suddenly persist for months or years, disappear and recur again. Subjective symptoms are absent.

The *Mycobacterium tuberculosis* may

Mycobacterium tuberculosis. The lesions consist of red soft irregular masses arranged in plaque formation. Lesions may or may not ulcerate.

The usual sites of predilection are the face, scalp, anal and perianal regions.



Fig. 608. *Framboesiforme Tuberculosis Cutis*. In a Negro.

Tuberculosis vegetans et frambiose may be secondary to glandular, bone and joint infection.

Diagnosis. *Tuberculosis vegetans et frambiose* is not to be confused with *sarcoma*. Subcutaneous injections of old tuberculin and a histologic study will clear up any confusion as to the diagnosis and will also rule out *sarcoma*.



Fig. 609. *Framboesiforme Tuberculosis Cutis*. In a Negro.



Fig. 610. *Framboesiforme Tuberculosis Cutis*. In Negro.

Miliary Tuberculosis

Cutaneous miliary tuberculosis, or *lichen scrofulosorum*, is very rare. It usually occurs among infants and children up to the age of puberty in whom it is a part of general miliary tubercu-



Fig. 613 Lichen Scrofulaceorum.



Fig. 612; Lichen Scrofulosorum.



Fig. 613 Lichen Scrofulaceus.

be demonstrated in the lesions or a positive reaction local general and even focal to tuberculin is usually present. Characteristic tubercle formation is found histologically. Ground tissue inoculated into guinea pigs has caused tuberculosis in some cases.

dissimilar conditions produced by lacillary embolism in connection with visceral lesions of tuberculosis or the dissemination of tuberculous antigens by the blood stream. Cutaneous reactions to these emboli vary greatly according to the individual's sensitivity. The lesions are



Fig. 614: *Lichen Scrofulosorum*. Left: Atypical tuberculous leg ulcers, developed during course of disease. Right: Histologic appearance of leg ulcer showing typical tuberculous architecture.

The disease often terminates in fatal tuberculous meningitis.

Diagnosis. *Lichen scrofulosorum* must be differentiated from lichen planus, follicular seborrheic dermatitis, dermatomyces, lichen spinulosus, and lichenoid syphilid. The tuberculin test and the presence of tuberculosis—either glandular, osseous, or other forms of cutaneous tuberculosis—are diagnostic features and are pathognomonic.

Tuberculids

SYNONYMS: *Toxotuberculids* (Hallopeau), *paratuberculoid* (Johnson).

Darier introduced the term "tuberculid" for a number of apparently very

bilateral and symmetrical. There are several varieties which have definite distinguishing characteristics. In connection with the tuberculids, it is important to remember that a diagnosis of cutaneous tuberculosis on a histopathologic study is only justified if typical tubercle formation with varying amounts of central caseation and zones of epithelioid cells, lymphocytes, plasma cells, and giant cells (Langhans type) are present, plus the demonstration of the Koch bacillus in the tissue or by animal inoculation. Collections of epithelioid cells and some giant cells may mean tuberculosis, but such collections also occur in other granulomata (leprosy, syphilis, etc.).

Kvile on the other hand believed that



Fig. 615 Papulonecrotic Tuberculosis. Note lesions on dorsa of hands as well as on knees and legs; lesions were also present on forearms.

the *Mycobacterium tuberculosis* could produce a simple nonspecific inflammatory reaction under certain still unknown conditions. In such instances prolonged observation may be required unless other evidences of tuberculosis are present (such as positive guinea pig

golgupoid granuloma annulare; lichen nitidus erythema elevatum diutinum.

Papulonecrotic Tuberculid

The commonest example of tuberculid is known as papulonecrotic tuberculid. It appears in successive crops on the extensor surfaces of the extremities. The eruption is due to the dissemination of toxins by the blood stream. Lesions vary in size but are always small. They begin as tiny papules, which undergo central necrosis, sloughing and crusting. Lesions eventually heal with the production of oval, sharply defined scars. The scars are white, atrophic, and slightly depressed. Itching is absent. Its absence distinguishes papulonecrotic tuberculid from prurigo. This variety occurs more frequently in adults between twenty and thirty years of age.

Acne and Acne Agminata

These terms are applied to a type of tuberculid localized on the face. The lesions appear suddenly—usually on the face, cheeks, chin, forehead, and eyelids—as firm red, deeply seated, isolated, and painless nodules. They gradually enlarge and in a few days are seen as pinhead and slightly larger sized conical or hemispherical dull red elevations, often with a central minute pustule. Their nodular element is easily palpable. Soon they tend to flatten, then disappear leaving a small depressed pigmented cicatrix. The eruption, as a rule, appears in crops over a period of four to six weeks, so that lesions in various stages of development and regression may be seen. The lesions vary in number from fifteen to twenty up to several hundred. There are no subjective symptoms. Complete recovery generally requires six to twelve months, and recur-



Fig. 616. Papulonecrotic Tuberculid. Showing some active lesions and numerous, old, pitted, pea-sized cicatrices on the extensor surface of forearm.

inoculation positive tuberculin test or skiagraphic findings in the lung). A focal reaction to tuberculin in the suspected lesion is highly suggestive.

Varieties. The following tuberculids are recognized: papulonecrotic tuberculid; acne (acne agminata); folliculoroseacea like tuberculid (Lewandowsky); acne scrofulosorum; erythema induratum; sarcoid of Boeck; sarcoidosis; Darier-Roussy sarcoid; lupus pernio; an-

rences are rare. It is not always easy to differentiate acutis from lupus miliaris disseminatus faciei in which the lesions have a typical apple-jelly color typical histologic structure of tuberculosis, and

Folliculitis

This is a type of papulonecrotic tuberculid characterized by the occurrence of firm, discrete, pinhead to pea-sized subcutaneous nodules on the hands, fore-



Fig. 617 Acutis. Some fifteen to twenty lesions of six months duration scattered over the face of fifty-year-old patient. Nose with lesions, especially on lower eyelids, to either side of nose and on chin, and healed lesions, as pigmented, slightly atrophic scars on forehead. Tuberculin patch test negative.

usually the *Mycobacterium tuberculosis*. The lesions in acutis are papulonecrotic and the picture histologically is that of an ordinary inflammation.

arms, feet, and legs. The lesions reach the surface in the course of days or a few weeks and become necrotic. They eventually heal, leaving atrophic scars.

The Rosacea-like Tuberculid of Leicaudourel

This lesion has a predilection for the cheeks, forehead and chin. The midface area and the nose are usually free from the disease. Lesions are characterized by

erupted from rosacea, milium lupus vulgaris, the small papular disseminated sarcoid of Boeck, and lupus pernio. Pustules are usually absent in rosacea-like tuberculid the color is yellowish rather than brownish and it does not disap-



Fig 618: Erythema Induratum. Left Whitfield type Right Ulceral type of Bazin.

bright red and brownish yellow discrete flat or slightly elevated pinpoint and slightly larger few or many papules which may appear follicular. Erythema scaling and telangiectasis are common symptoms. A few pinpoint pustules are occasionally present. There is generally marked sensitivity to the tuberculin test. Rosacea like tuberculid must be differ-

entiated from rosacea, milium lupus vulgaris, the small papular disseminated sarcoid of Boeck, and lupus pernio. Pustules are usually absent in rosacea-like tuberculid the color is yellowish rather than brownish and it does not disappear completely under vitro-pressure. Rosacea like tuberculid does not as a rule respond well under treatment designed for rosacea but does well under tuberculin and gold sodium thiosulfate injections. There are cases, however in which the differential diagnosis is not possible. The lesions of Boeck's sarcoid are more deeply seated and are larger. Lupus pernio is

volves other areas and the patient's skin in lupus pernio and sarcoid gives a negative tuberculin test. However the tuberculin test is occasionally negative in the Lewandowsky tuberculid.

Acne Scrofulosorum

This lesion is probably a transition form from lichen scrofulosorum to papulonecrotic tuberculid. Lesions appear as discrete, pinhead-sized, acuminate, reddish papules involving the hair follicles. The sites of predilection are the buttocks and external aspect of the limbs. The lesions undergo rapid pustulation and crust formation. Funnel-shaped depressed scars follow healing. The condition occurs in scrofulous patients and has no subjective symptoms.

Erythema Induratum

SYNONYMS *Erythema induratum scrofulosorum*, *Bazin's disease*, *erythema induratum scrofulosum* (*Bazin*)

Erythema induratum is a chronic recurring disease seen commonly on the calves of the legs among girls and young women whose occupations keep them on their feet the greater part of the day. It is occasionally seen in boys and men. It is characterized by variously sized, subcutaneous, tender but not painful nodules which terminate in resolution or necrosis. The lesions assume bluish-red or violaceous-red color.

The nodules are hard and are discovered by palpation when not large enough to become visible. They may vary in diameter from $\frac{1}{2}$ to 1 or more inches. In some cases nodules and ulcers recur every winter. The open lesions may be very superficial and simulate ecthyma or ulcers.

However ulcers resulting from necrosis are usually deeply excavated, ir-

regularly shaped, and have overhanging, undermined borders. The histopathologic findings are not always characteristic and vary from typical tubercle formation to nonspecific changes and fibrosis.

The tuberculin test is positive in a large percentage of the patients.



Fig. 619 *Erythema Induratum*, Ulcerative form of *Bazin*.

Erythema induratum is distinguished from *erythema nodosum* by its chronic course, the tendency to ulceration, the absence of pain, the presence of scars, and its frequent association with tuberculosis. It is differentiated from *gummatous syphiloderm* by the symmetry of lesions and by the absence of concomitant signs of syphilis.

Sarcoid of Boeck

SYNONYMS *Multiple benign sarcoid* (*Boeck*) *benign nodular lupoid* (*Boeck*) *nodular lupoid* (*Darier*)

The term "sarcoid" was introduced by Kaposi to identify sarcoma-like neo-



Fig. 620: Sarcoids. Mill size papules on neck and face



Fig. 622: Sarcoid. Purplish-red, large pea-sized lesion with surface telangiectases. Eight weeks duration. Disappeared under radiation with x rays, 1½ E.D. week (five exposures) and neoarsphenamin intravenous



Fig. 621: Sarcoids. Limited to rim of nares.



Fig. 623: Dermohypodermic Sarcoids. Of three years duration.



Fig. 621 Multiple Sarcoids.

plasma. Sarcoid is characterized by grouped or discrete variously sized (lentil seed to grape and larger) cutaneous or subcutaneous, nodules single or multiple diffusely infiltrating plaques, and gyrate lesions, running a benign course and possessing a tuberculous histopathology. Sarcoid is a disease of adults commonly seen among females. The sites of predilection are the face suprascapular regions, and extensor surfaces of the arms.

Nodules of sarcoid are round, firm, elastic, and assume a purplish hue. They do not ulcerate or recur after removal and they do not metastasize.

The plaques of sarcoid are similar to nodular sarcoid, and involve the whole thickness of the skin without any attachment to underlying structures.

Lesions tend to involute in the center spread peripherally in raised ring formation and heal without scarring.



Fig. 623 Multiple Sarcoids.



Fig. 626: Multiple Sarcoidosis. (Courtesy of Dr. C. C. Thomas.)



Fig. 627: Sarcoidosis. Infiltrative purplish-red plaque on bridge of nose and cheeks simulating lupus erythematosus. Moderate dactylitis left ring and right little finger due to osteolytic changes in phalanges (see diagram, Fig. 629). Complicating verrucae on right palm. Both ear lobes were enlarged and purplish red. (Courtesy of Dr. Arthur Finklestein.)

Face lesions are disfiguring because they become blue in the center and yellow at the border.

Darier and Rouxy believed sarcoids to be due to some low grade infection by the B. tuberculosis. They based their opinion on the histopathology and the perivascular lymph origin of sarcoids. However the tuberculin test is generally negative as is guinea-pig inoculation with ground portions of affected tissue.

It is likely that sarcoids represent special types of cutaneous reaction capable of being produced by a variety of agents, among them tuberculosis, leprosy, syphilis, and it is also a disease in a class by itself.

Sarcoids are differentiated from *leu kerma cutis nodular lupus erythematosus*, and *erythema induratum* by the symptomatology and the characteristic tuberculous histopathology.

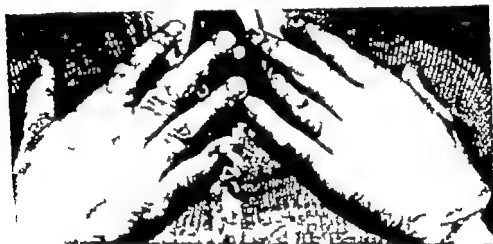


FIG. 628 Sarcoidosis. Note diffuse slightly infiltrated, congestive redness of the fingers. Also note shortening of left ring and right little fingers due to absorptive changes in phalanges (skiagraphic). There was infiltrative enlargement of lobe of right ear. (Courtesy of Dr. Arthur Fleischman.)

Sarcoidosis

SYNONYMS *Hutchinson-Breuer*, *Borch-Schaumann syndrome*, *benign lymphogranulomatosis*, *cryptic histiocytosis*, *respiratory fever of Herfordt*, *acnaloglandular syndrome (Parinaud)*, *sarcoid tuberculosis multiplex cystica*.

These terms refer to localized manifestation of the same granulomatous, generalized disease. In this syndrome, or protean disease the microscopic picture is the same irrespective of the localization of the active lesions which may be in the skin (sarcoid and lupus pernio)

in the lungs, lymph nodes, spleen, liver bones, salivary glands, or eyes—in part or in all of these structures in a given patient at one and the same period of its evolution. Although classified under tuberculosis, it is not, by many believed to be of tuberculous etiology. However a diagnosis of sarcoidosis in the Negro must be made with reserve, since a number of such cases have eventuated in fatal miliary tuberculosis. Furthermore, many instances of sarcoid in the Negro appear to be attenuated forms of cutaneous tuberculosis.



Fig. 626: Multiple Sarcoids. (Courtesy of Dr. C. C. Thomas.)



Fig. 627: Sarcoidosis. Infiltrate: purplish-red plaques over bridge of nose and cheeks simulating lupus erythematosus. Moderate dactylitis: left ring and right little finger due to osteolytic changes in phalanges (see diagram Fig. 629). Complicating erythema on right palm. Both ear lobes were enlarged and purplish red. (Courtesy of Dr. Arthur Finklestein.)

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Darier and Roussier believed sarcoids to be due to some low grade infection by the B tuberculosis. They based their opinion on the histopathology and the perivascular lymph origin of sarcoids. However the tuberculin test is generally negative as is guinea-pig inoculation with ground portions of affected tissue.

It is likely that sarcoids represent special types of cutaneous reaction capable of being produced by a variety of agents, among them tuberculosis, leprosy, syphilis, and it is also a disease in a class by itself.

Sarcoids are differentiated from *leukemia cutis nodularis*, *lupus erythematosus* and *erythema induratum* by the symptomatology and the characteristic tuberculous histopathology.

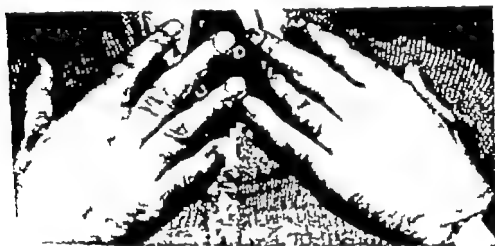


Fig. 628 Sarcoidosis. Note diffuse, lightly infiltrated, congestive redness of the fingers. Also note shortening of left ring and right little fingers due to absorptive changes in phalanges (skiographic). There was infiltrative enlargement of lobe of right ear. (Courtesy of Dr. Arthur Fleckenstein.)

Sarcoidosis

SYNONYMS *Hist. Hansen-Brounker-Borch-Schimmann syndrome, bovine lymphogranulomatosis, atypical trichocytitis, arthropod fever of Heerfordt, oculoglandular syndrome (Parinaud), ovoids tuberculae, multiple cysts.*

These terms refer to localized manifestations of the same granulomatous, generalized disease. In this syndrome, or protean disease, the microscopic picture is the same irrespective of the localization of the active lesions which may be in the skin (sarcoid and lupus pernio)

in the lungs, lymph nodes, spleen, liver, bones, salivary glands, or eyes—in part or in all of these structures in a given patient at one and the same period of its evolution. Although classified under tuberculosis, it is not, by many, believed to be of tuberculous etiology. However a diagnosis of sarcoidosis in the Negro must be made with reserve, since a number of such cases have eventuated in fatal miliary tuberculosis. Furthermore, many instances of sarcoid in the Negro appear to be attenuated forms of cutaneous tuberculosis.

In the majority of cases, the tuberculin test is negative and animal inoculations of tissue fail to cause tuberculosis. By some this is thought to be the result of a state of anergy to the chemical products of the Koch bacillus.

The clinical manifestations in sarcoidosis include the cutaneous and ocular le-

both the nodular and indurative types.

1 Nodular Darier Roussy sarcoids appear on the limbs and face vary in size from a pea to a cherry and are deep red as they approach the surface.

2 Indurative Darier Roussy sarcoids feel somewhat like hard movable nodes in the subcutaneous tissue appear on

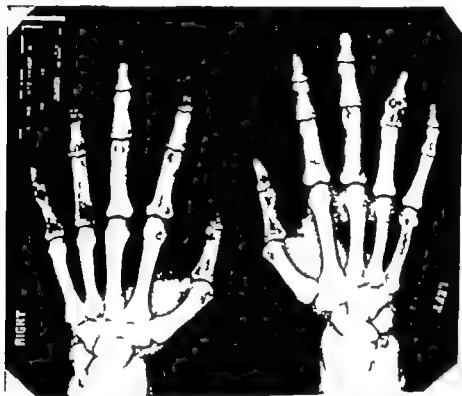


Fig. 629: Sarcoidosis. Note the rarefied or cystic-like radiolucent areas in many of the phalanges.

sions, a Mikulicz like syndrome, generalized lymphadenopathy, splenomegaly and lesions in the lungs and bones (fingers, with brawny swelling of one or several of these hands, feet, and skull) revealed skiagraphically as rarefied areas. Hyperglobulinemia is not an infrequent finding. Absolute diagnosis is made by finding the typical, microscopic, pathologic changes.

Darier Roussy Sarcoids

Darier Roussy sarcoids originate in connection with the cutis and are of

the thighs and buttocks during adult and advanced life. The condition may however occur anywhere on the body.

Treatment Sarcoids and sarcoidosis are amenable to a variety of agents. For cutaneous lesions, the best local therapy is *x ray treatment* (5 r) at weekly intervals for eight exposures. *Heavy metal, systemic therapy* (arsenic, intramuscular injections of bismuth and gold salts intravenously) has been successfully used. *Ethyl chaulmoograte* (antileprol) is of value in sarcoidosis, but the disease has a tendency to spontaneous remission.

Lupus Pernio (Hutchinson)

Chilblain lupus of Hutchinson is in reality the cutaneous manifestation of sarcoidosis.

Lupus pernio is accompanied by vivid red or purplish areas of congestion on the face, nose, ears, hands, and feet. The lesions are symmetric, demarcated, infiltrated, soft, and often marked with telangiectasia. Red violaceous, swollen ear lobes are present at times. Diacopic examination reveals minute tubercles. The disease is sometimes associated with lupus vulgaris.

Lupus pernio differs from true chilblains by persisting throughout the summer.

The histology is identical to sarcoid. It is not an active tuberculous process.

Angiolupoid

Angiolupoid is a rare, tumorlike growth, possessing tuberculous features and numerous dilated blood vessels.

Granuloma Annulare

SYNONYM *Ringed eczema*.

Granuloma annulare is a chronic inflammatory disease characterized by the presence of whitish nodules within the dermis. The nodules spread gradually to form circular lesions. The typical lesion, therefore, is annular with a clear center. Atypical lesions alone, and in association with typical ones, are observed, consisting of discrete small, flattened nodules and plaques, the result of confluence of such papules and nodules.

Etiology The cause is unknown. The Koch bacillus has been found in an occasional case. Reaction to tuberculin is variable.

Some clinicians class it among the lichen group.

Pathology The histopathology of

granuloma annulare is confined to the corium. Early lesions show a dense infiltration consisting of lymphocytes, polymorphonuclear cells, epithelioids, and numerous spindle cells. There is a granular degeneration of the collagen bundles with a cellular reaction around this. Mast



Fig. 636 Granuloma Annulare.

cells are occasionally numerous and giant cells are rarely present. Local edema is always present.

Clinical Features The primary lesions consist of few pink or white, minute nodules within the dermis extending peripherally and forming small plaques. Resolution begins in the center of lesions, leaving slightly discolored areas which do not undergo atrophy and scar formation. Coalescence of adjoining lesions may lead to gyrate figure formation.

The disease is common among children but may occur in young adults. Lesions occur on the dorsum of hands and feet, although any part of the body may be involved. The disease starts

usually in summer and may persist for years. It may disappear spontaneously without ulceration and secondary infection. The tendency in general is for the lesions eventually to disappear spontaneously. The lesions often disappear follow-

mucin are positive in granuloma annulare and negative in necrobiosis lipoidica diabetorum.

Erythema Elevatum Diuturnum

The cause of this is unknown; it is, however, believed to be a variety of *granuloma annulare*.

It is characterized by firm, painless nodules varying in size from 1 to 3 cm. in diameter. At first they have a reddish color which gradually becomes purplish. These nodules tend to coalesce and form irregular shaped elevated plaques. The sites of predilection are the extensor surface of limbs and joints, although they



Fig. 631. Granuloma Annulare. On upper lip. Unusual location.

ing the removal of a section for histologic study.

Diagnosis. The morphologic picture is characteristic. Annular sarcoid is differentiated by the histology. In necrobiosis lipoidica diabetorum there are nonelevated plaques with a reddish periphery and yellowish center and histologically there are lipid deposits interspaced between the connective tissue bundles. These extracellular fat deposits are rarely found in granuloma annulare. According to Montgomery, stains for



Fig. 632. Granuloma Annulare. Dorsum of right foot. Ten months' duration. Disappeared under x-ray therapy.

may occur anywhere on the body surface. The disease runs a course similar to granuloma annulare and the treatment is that of granuloma annulare.

Lichen Nitidus

This variety is characterized by the appearance of small, flat, glistening round or polygonal papules which have the same color as the skin or a yellowish brown hue. They often show a central depression. The sites of predilection are the penis, the abdomen, the flexor surface of the arms, and the palma. Subjective symptoms are generally absent, a mild pruritus occurs occasionally. The mucosa of the mouth is occasionally involved.

The lesions do not resolve and continue unchanged. The etiology is unknown. However it is considered, by some a variant of lichen planus, since it has been observed to improve under arsenical therapy intramuscular injections of bismuth salicylate and under gold sodium thioarsenate intravenously.

It must be differentiated from lichen planus and from verruca planum. Itching is present and the color more violaceous in lichen planus. The pathology of warts is entirely different from that of lichen nitidus.

The histology of lichen nitidus is similar to that of an inflammatory granuloma although some lesions show a tuberculous architecture.

Lupus Erythematosus

SYNONYMS *Lupus sebaceus, orbicularis congestiva (Hebra) alerythema centrifugum (Unna) Lupus erythematosus.*

Lupus erythematosus is an inflammation of the skin characterized by the presence of erythematous, usually scaly defined patches of various sizes and shapes. It involves a definite area, usually of the nose, cheeks, ears, dorsa of hands, scalp, and mucous membrane of the mouth.

Etiology The cause of lupus ery-

thematosus is not known. It is a disease of the body as a whole—a constitutional affection. The skin lesions may be and frequently are the only manifestations of the disease. They appear spontaneously or are precipitated by sunlight or



Fig. 633 *Lupus Erythematosus*. Of nose and vermillion border of upper lip. Numerous ephelides are also present.

trauma. Systemic disturbances may develop coincidentally with, or long after the skin lesions.

The oldest theory of its causation is that it is produced by the Koch bacillus or its toxins. The tendency now, however, is to disregard this viewpoint. Others believe it is due to streptococci, to sensitization to the products of some organism, or to metabolites. Finally by some, it is believed to be a virus disease. The allergic theory (Teilum) rests upon the fact that the disease affects the collagenous tissue and the changes in the skin and small vessels of the viscera often lead to fibrinoid degeneration, a condition also seen in allergic diseases. However such connective tissue changes are also found in a variety of nonallergic diseases.

Among the known precipitating factors in acute and subacute cases is excessive exposure to sunlight or ultraviolet

rays from artificial sources. Persons with chronic, cutaneous forms of the disease have, however, been observed to spend hours in the sunlight without harm.



Fig. 634. Lupus Erythematosus Disseminatus.

Varieties. The following classification appears clinically practical:

- I. Acute disseminated lupus erythematosus.
- II. Subacute disseminated lupus erythematosus (Libman-Sachs syndrome).
- III. Chronic lupus erythematosus. This is the common form and three varieties are distinguishable:
 - (1) Symmetrical centrifugal erythema (Biett).
 - (2) Fixed or discoid lupus erythematosus.
 - (3) Lupus erythematosus profundus.

The terms "acute" and "subacute" commonly designate those presenting both cutaneous and systemic disturbances. If recovery occurs, the skin lesions may recur from time to time with or

without systemic disturbances. The cutaneous lesions in both acute and subacute are very superficial and apt to be purely erythematous. The term chronic generally designates a persistent cutaneous form without systemic disturbances. The term "disseminated" refers to both



Fig. 635: Acute Disseminated Lupus Erythematosus. Recurrent over a period of seven years. Present attack followed exposure to sunlight. Had small patch on face for several months. This patient had three previous attacks of the disease limited to her face all of which disappeared spontaneously. A previous attack had been as severe as the present one and followed prolonged exposure to the sun while berry picking. The present attack was fatal.

the extent of the cutaneous involvement as well as to the degree of systemic disturbance. However extensive and disseminated skin lesions may be present

with but mild or no systemic disturbances and vice versa. In chronic forms, dissemination, or rather many new lesions, may appear suddenly over the skin, but it is rarely accompanied by systemic disturbances. In this secondary acute type constitutional disturbances are not so

vent of the cases, in young girls or women who usually do, but may not, show cutaneous lesions. The onset is ordinarily sudden with marked fever transient, persistent, or recurrent, faint or marked erythematous to violaceous, usually non-scaly macules, generally appearing first on the face over the bridge of the nose and the cheeks. The lesions at first are few in number and discrete, later discrete but numerous, or the macules may coalesce to form large erythematous patches. In some the skin of the entire face, neck, and upper part of the body may become violaceous-red with slight or moderately marked edema (erysipelas peristans faciei of Kaposi or lupus erythematosus exanthematicus). A mild eruption is no criterion of the seriousness or severity of the visceral lesions. Serious constitutional disturbances, associated with a variable degree of joint, muscle and back pains may be present. The disturbances in the viscera may precede accompany or follow visible cutaneous lesions. If visceral disturbances precede the clinical diagnosis may be suspected, but only becomes definite when the skin lesions appear. Reversal of the albumin-globulin ratio, leukopenia, and an increased red-cell sedimentation rate indicate the degree of systemic involvement. Albuminuria is not present in all cases but is serious when it is present. A fatal outcome, which is common in several weeks to several months, often occurs in a patient mentally clear to the end. In some the fatal outcome, in the absence of any systemic, discoverable alterations other than the blood changes noted, is explained only on the basis of an intense toxemia.

Repeated examinations of the renal and blood functions are indicated in all instances of acute lupus erythematosus no matter how mild the cutaneous phenomena although the site and general



Fig. 636 Acute Exanthematic Lupus Erythematosus. There was marked anorexia. Recovery. Subsequent history not available.

severe as in the primary acute types. The term disseminate to indicate systemic dissemination of the skin lesions, is an erroneous concept of the disease since it is systemic from the outset.

Acute Disseminated Lupus Erythematosus. The acute and subacute forms are uncommon but serious. They differ partly in the systemic disturbances present and largely in the prognostic outlook. They may pass insensibly into one another.

The acute form occurs, in about 25 per

appearance of the erythema rather than its course may be its chief characteristic. The duration of the erythema varies, in some the temperature of the patient may be but slightly elevated and the systemic lesions despite absence of subjective signs may be severe especially of renal

the superficial chronic forms noted below

Subacute Lupus Erythematosus. This form is similar to the acute, but the objective and subjective signs, at least in the beginning are less marked. A fatal termination eventually occurs, however in probably 50 per cent of the cases. A



Fig. 637: Lupus Erythematosus Disseminatus. Subacute form.

and blood elements. These types are usually seen by the general practitioner and are often unrecognized because the eruption may be mild, or transient and the systemic disturbances, often severe may even precede the onset of the dermatosis. Recovery may be followed by relapse once or several times, ending finally in death or it may be followed by one of

duration of six to twelve months is common with fatal termination or recovery and later relapse. The onset is insidious and may be noncontaneous, or the cutaneous lesions may be transient. Several weeks to several months of weakness, fatigue, mild muscle pains, especially of the thighs and legs, and joint pains may precede. Slight fever which is long-con-

trued and constant but irregular with periods of exacerbation, precedes or accompanies telltale nephritic, urinary findings. Progressive injury to the renal vascular system with damage to the renal parenchyma leads to azotemia. At a given point, violaceous-red or erythematous spots, which may persist, disappear and recur and which may or may not ever appear generally on the face, or

affected membranes, a remittent, cachectic course with loss of weight, and central nervous system disturbances. Reversal of the serum-albumin-globulin ratio with the globulin increased is usual (Tauszig). Red-cell sedimentation rate is accentuated to a varying degree; it is constant, however (Coburn and Moore). In both acute and subacute lupus erythematosus, the blood picture should be



Fig. 633. Lupus Erythematosus Dissemminatus. Subacute form.

face and extremities. The skin lesions, which may be violaceous and show punctate hemorrhages, usually appear first on the nose and cheeks, then neck, trunk, and extremities. The diagnosis becomes certain only with the appearance of the characteristic cutaneous lesions. Criteria for the diagnosis of acute and subacute lupus erythematosus are not always clear cut. All or some of the following are persistently present or occur at some time during the course of the disease: arthralgias, pleuropulmonary disturbances, anorexia, progressive anemia, leukopenia, progressive anthesis, thrombocytopenia, nephritis associated with edema and seroplastic exudates, lymphadenitis with pain over the

regularly checked for progressive anemia and for total and fractionated blood protein—the latter especially in those with albuminuria. In the presence of an albuminuria, the renal concentration and urea-clearance tests are the best indexes of early depreciation of renal functional reserve. Even minor degrees of anemia are significant. Biologically false Wassermann tests have occasionally been found believed by some to be due to the increase in globulin. Blood cultures are negative. In analyzing the post-mortem renal findings in fifteen cases of acute and subacute lupus erythematosus, Stickney and Keith concluded that a common, distinct renal lesion does not exist, and that the renal changes probably re-

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affected membranes, a remittent, cachectic course with loss of weight, and central nervous system disturbances. Reversal of the serum-albumin-globulin ratio with the globulin increased is usual (Tausig). Red-cell sedimentation rate is accentuated to a varying degree; it is constant, however (Coburn and Moore).

In both acute and subacute lupus erythematosus, the blood picture should be



Fig. 633. Lupus Erythematosus Dissemminatus. Subacute form.

face and extremities. The skin lesions, which may be violaceous and show punctate hemorrhages, usually appear first on the nose and cheeks, then neck, trunk, and extremities. The diagnosis becomes certain only with the appearance of the characteristic cutaneous lesions. Criteria for the diagnosis of acute and subacute lupus erythematosus are not always clear cut. All or some of the following are persistently present or occur at some time during the course of the disease: arthralgias, pleuropulmonary disturbances, anorexia, progressive anemia, leukopenia, progressive asthenia, thrombocytopenia, nephritis associated with edema and seroplastic exudates, alopecia, polyserositis with pain over the

regularly checked for progressive anemia and for total and fractionated blood protein—the latter especially in those with albuminuria. In the presence of an albuminuria, the renal concentration and urea-clearance tests are the best indexes of early depreciation of renal functional reserve. Even minor degrees of anemia are significant. Biologically false Wassermann tests have occasionally been found believed by some to be due to the increase in globulin. Blood cultures are negative. In analyzing the post mortem renal findings in fifteen cases of acute and subacute lupus erythematosus, Stekney and Keith concluded that a common, distinct, renal lesion does not exist, and that the renal changes probably re-

appearance of the erythema rather than its course may be its chief characteristic. The duration of the erythema varies in some the temperature of the patient may be but slightly elevated and the systemic lesions, despite absence of subjective signs, may be severe especially of renal

the superficial chronic forms noted below

Subacute Lupus Erythematosus This form is similar to the acute, but the objective and subjective signs, at least in the beginning are less marked. A fatal termination eventually occurs, however in probably 50 per cent of the cases. A



Fig 637: Lupus Erythematosus Disseminatus. Subacute form.

and blood elements. These types are usually seen by the general practitioner and are often unrecognized because the eruption may be mild, or transient, and the systemic disturbances, often severe may even precede the onset of the dermatosis. Recovery may be followed by relapse once or several times, ending finally in death or it may be followed by one of

duration of six to twelve months is common with fatal termination or recovery and later relapse. The onset is insidious and may be noncutaneous, or the cutaneous lesions may be transient. Several weeks to several months of weakness, fatigue mild muscle pains, especially of the thighs and legs, and joint pains may precede. Slight fever which is long-con



Fig. 640 Discoid Lupus Erythematosus.



Fig. 641 Fixed Lupus Erythematosus. Left On bridge of nose and upper lip. Of eight months duration. Right In five-year-old. Improved during the winter. *Sunlight always precipitated severe outbreak.

sult from a toxic process and not from primary renal disease. Proliferative and thrombotic lesions occur on the valves of the heart in at least 20 per cent of the cases (Baehr).

Chronic Lupus Erythematosus This is the common form.

SYMMETRICAL CENTRIFUGAL ERYTHEMA This begins as a varying sized

atrophic area will mark the site. Those with a rapid course may develop into the discoid form. In some there is a varying degree of fine sometimes thick, more or less adherent scaling on the under surface of which may be found characteristic keratotic plugs. Rosacea, seborrheic dermatitis, and epithelioma may be simulated.



FIG. 639 *Left: Lupus Vulgaris Erythematosoides. Note butterfly distribution. Gland pressure showed many "apple-jelly" nodules. Right: Lupus Erythematosus (symmetrical centrifugal erythema (Biett). Duration two years. Typical butterfly distribution.*

area of congestive redness, often marked by telangiectases, and usually over the bridge of the nose or cheek structures, the ears, scalp and fingers. This purely erythematous or telangiectatic type is very superficial, often symmetrical, and has a well-defined slightly elevated border and a slightly depressed center. There is a marked tendency to centrifugal extension with central clearing. The patch is sensitive to palpation. The onset is more or less sudden, but it often disappears and reappears, commonly without a trace of where it had been. It may however remain stationary for long periods, and then, as it disappears, a faint

FIXED OR DISCOID LUPUS ERYTHEMATOSUS Discoid lupus erythematosus is the usual form of the disease. It may develop from the preceding form. It occurs in both sexes, with an increased incidence among females in the ratio of 5:1. The disease begins in the third decade of life in over 40 per cent of cases and occurs between the ages of twenty and thirty years in 25 per cent of patients. It is rare among children.

An evidence of gland, bone, or joint tuberculosis is usually present in 80 per cent of cases. Pulmonary tuberculosis is rarely seen in conjunction with lupus erythematosus.

scars of dull white tint are left by these uniform and superficial lesions. The scars are never pigmented, puckered, radiated, corded, or deeply attached.

Discoid lupus erythematosus can be readily differentiated from seborrheic dermatitis, acne rosacea and lupus vulgaris by the history of the disease, the presence of patulous follicles and epithelial plugs on the under surface of the adherent scales, and the tendency to central atrophy.

LUPUS ERYTHEMATOSUS PROFUNDUS

This deep form was originally described by Brocq as consisting of deep-seated, tumor like lesions usually associated with typical lesions of lupus erythematosus. The lesions closely resemble the Darier Roussy sarcomatoid or the Spiegler Fendt sarcomatoid. Histologic examination commonly shows keratotic plugging of the follicular and sweat-duct orifices.

Prognosis in Cutaneous Tuberculosis

This varies in the different varieties of cutaneous tuberculosis. In *lupus vulgaris* the duration of life is not affected, although disfigurement is the rule. If the disease develops in midlife, the prognosis for a cure is better than if it develops during childhood.

The prognosis in *lupus nodulosus* is excellent, and a cure usually follows proper treatment.

The prognosis in *scrofuloderma* is guardedly favorable.

The prognosis in *tuberculosis ulc* is very unfavorable.

The prognosis in generalized *miliary tuberculosis* is unfavorable.

In *lupus erythematosus* the chronic discoid type offers a good prognosis, although recurrences are common. Death occurs in the majority of cases of acute

and in a lesser percentage of the subacute forms of lupus erythematosus.

The prognosis in the other varieties is favorable except in *tuberculosis* in this condition recurrences are common.

Treatment in Cutaneous Tuberculosis

The routine treatment for pulmonary tuberculosis is applied equally well to patients suffering from cutaneous tuberculosis and the tuberculids. Measures are taken to increase the patient's resistance. These consist of maximum exposures to sunlight outdoor exercise, and a well balanced nutritive diet. It is advantageous to improve the patient's environment by making a change in residence and climate. There is no specific internal medication for treating cutaneous tuberculosis. Cod liver oil, vitamins A and D hypophosphites, potassium iodide syrup of iron iodide urea chloride and thyroid extract are useful in treating cutaneous tuberculosis.

According to Charpy lupus vulgaris has almost completely disappeared in France since the introduction of vitamin D₂ (dihydrocholiferol) in its therapy. His method consists in giving 15 mg (600,000 international units) in an alcoholic solution by mouth weekly for three months, then every two weeks for three months, and after that monthly during the winter months. Since over dosage may cause nausea, vomiting, diarrhea, anorexia, polyuria, and even calcification of the soft tissues, renal tubules, and blood vessels, a guide to the proper dosage should be the symptoms rather than estimations of blood calcium. If a focal reaction occurs with the first few doses, the drug may be stopped and later resumed. The minimum effective dose appears to be 50,000 unit daily.

The lesions are erythematous and follicular asymmetrical and usually localized to one area of the face scalp and mucosae. Discoid lupus erythematosus consists of variously sized flat well defined persistent spots, ranging in color from red to violet and covered with adherent grayish yellow scales. The spots

It frequently attacks the scalp where it starts as red scaly patches which terminate in smooth scars. The patches on the scalp remain hairless and are surrounded by narrow scaly areas. Intolerable itching of the scalp is not infrequent. The dorsa of hands and fingers are occasionally involved with lesions resembling



Fig. 612 Fixed Lupus Erythematosus. Left: Extensive Right: With a superimposed dermatitis from intra-lesionally injected gold sodium thio-sulfate

do not fade away under pressure. The scales are occasionally oily. The lesions may be slightly elevated above the skin level. Vesicles and bullae rarely occur in discoid lupus erythematosus. The patulous sebaceous, gland-duct orifices, orifices into which a variable number of keratinized plugs from the under surface of the adherent scales fit become evident when the scales are shed.

Discoid lupus erythematosus is slow in progress and may persist for years. Its chronicity is accompanied by central dermic sclerodermatophy of varying degree. Discoverable systemic disturbances are rare.

chilblains. Lesions of discoid lupus erythematosus do not appear in the warm weather.

Discoid lupus erythematosus involves the mucous membrane of the lips and oral mucosa in about 25 per cent of cases. Patches on the lips and mouth appear as areas of grayish hyperkeratosis, which undergo erosion and ulceration, and are encircled by violaceous inflammatory zones. Lip lesions resemble dried layers of collodion. Lesions of the buccal mucosa appear as asymmetrically arranged white spots surrounded with red margins. They may also become punctate. White

The local remedies for tuberculosis are legion and inadequate. Favorable results have been reported in lupus vulgaris from employing poultices of ordinary table salt. Poultices of a 1 per cent solution of mercury bichloride are beneficial. Refrigeration by solid carbon dioxide produces an intense reaction which is followed by a pliable scar. The treatment is painful, however, the cosmetic result is good.

Improvement followed scarifying lesions of lupus vulgaris and applying three times each week solutions of 30 per cent zinc chloride and 80 per cent alcohol. Bandages saturated in a solution of tincture of iodine and alcohol (tincture of iodine, 10 cc. to 90 per cent alcohol, 90 cc.) are applied in intervals between treatments.

Kile's method is a new treatment in lupus vulgaris. The procedure consists of the injection of a 15 per cent aqueous suspension of cornstarch into the lesions of lupus vulgaris. Several areas may be treated at one time. The procedure is not painful, but a reaction occurs in twenty-four hours at the site of injection, which consists of erythema, a papule, or a nodular swelling. The reaction slowly subsides. The starch is sterilized by baking at 150° C. in an oven. A 15 per cent suspension is prepared in a sterile rubber-stoppered vial, and only 10 cc. is made up at a time. Contamination is avoided by making fresh suspensions frequently. A tuberculin syringe with a 20- or 21-gauge needle is used. The amount given at each injection is 0.1 or 0.5 cc. and is not repeated for two weeks. The results of this therapy have been encouraging.

If the area of involvement is accessible and not too large, wide surgical excision is the treatment par excellence. Surgical diathermy is widely employed for the

destruction of the deep-seated nodules.

Pfannenstiel advised 0.5 gm. (7½ grains) of sodium iodide six times each day for treating intranasal lupus. The nasal chambers are thoroughly cleaned each morning with an antiseptic douche, dried and packed with sterile gauze saturated in a 2 per cent solution of hydrogen peroxide. This packing is left in situ and moistened from time to time by hydrogen peroxide carried by a pipet.

A powder consisting of equal parts of potassium iodide and chalk is insufflated over ulcerated areas of mucous membranes and subsequently washed with chlorine water. This chlorine water is prepared by adding potassium chlorate to a concentrated solution of hydrochloric acid and subsequently diluted in water.

Scrofuloderma should be treated by roentgen irradiation with filtered rays. Surgical excision is indicated where the primary tuberculous lesion is single; however, when the glands are matted together and ulceration is present, radiotherapy is preferable. Ultraviolet therapy is also beneficial.

Erythema Induratum. The local treatment of erythema induratum consists of irradiation by ultraviolet light. Andrews has obtained excellent results from gold injections and medical diathermy. Favorable results have been reported from tuberculin injections.

Granuloma Annulare. Granuloma annulare as a rule rapidly disappears after suberythema exposures of x-rays. Andrews advises doses of 200 r (½ E.D.) given unfiltered at intervals of two weeks with the skin surface shielded to within 1 inch of the border of the plaque. Gold sodium thioarsate or colloidal gold is also more or less specific therapy. Andrews recommends weekly injections of a 1 per cent solution be-

This drug is also effective in lupus verucosus

Dietotherapy In recent years, treatment by dietary measures has been advanced as a method of value in cutaneous tuberculosis. Hippocrates was the first to call attention to the benefits derived from diet in the healing of wounds. Struwe a century ago advocated a salt free diet for treating cutaneous tuberculosis.

Gerson championed dietotherapy for treating skin tuberculosis. Sauerbruch and Herrmannsdorfer advised a diet differing but slightly from that of Gerson's.

The Gerson diet is base forming while that of Sauerbruch and Herrmannsdorfer possesses an acid forming value. Von Noorden and others are of the opinion that the action of their diet is independent of acid or base-forming properties.

Gerson's diet excludes table salt, alcohol, tobacco, coffee, tea, water, soda, water, lemonade, all kinds of mineral water, soups, cream, condiments and spices (pepper, vinegar and mustard), canned food, preserved meats (salted, smoked or pickled), ham, sausage, sardines, salmon and caviar. He also prohibits chocolate, cocoa and all forms of cheese except cottage cheese.

Keimig and Hopf have recently modified dietotherapy. They rule out table salt from the diet, substituting for it an equilibrated mixture of salts containing sodium chloride, potassium, calcium, and magnesium salts.

The basic principles common to these special diets are lack of salt, inclusion of large quantities of fresh vegetables and fruits rich in vitamins, and alteration in the percentage of proteins, fats, and carbohydrates. These diets depend upon the benefits obtained from a liberal intake of fresh uncooked articles of food like fruits, vegetables, eggs, milk, and

minced meats. Gerson allows the yolk of 1 egg and 250 cc ($\frac{1}{2}$ pint) of milk each day while Herrmannsdorfer gives $1\frac{1}{4}$ quarts of milk. These diets presumably dehydrate the skin and alter its ionic index, thereby influencing body chemistry and making a less suitable soil for the growth of the *Mycobacterium tuberculosis*.

The exact status of diet as a method in the treatment of skin tuberculosis, and lupus vulgaris in particular is still undecided.

Tuberculin therapy has a place in treatment, and it is believed that it will gain a better place in the future, especially in the tuberculids. Tuberculin therapy is not indicated for cases with local congestion and in individuals with pulmonary tuberculosis. Tuberculin is administered subcutaneously in very small doses at long intervals to elicit a local reaction. Tuberculin does not immunize the person against tuberculosis; it immunizes him against tuberculin itself.

Phototherapy obtained by exposures to Finzen's light, the carbon-arc light, the quartz light and the so-called cold quartz lamp accomplishes the best results. Irradiation by a carbon-arc lamp is preferable because it approaches more closely the solar spectrum. Large doses of ultraviolet light are given to involved areas and smaller areas are applied to the rest of the body surface. The cold quartz lamp is indicated for mucous-membrane lesions. The course of treatment is continued over a period of six months after lesions of skin tuberculosis have apparently healed in order to prevent recurrences.

Irradiation by x rays and radium is of definite value in hypertrophic lupus vulgaris but harmful in atrophic forms of the disease.

gerous symptoms, such as purpura haemorrhagica, hepatitis jaundice, albuminuria metrorrhagia, pruritus, and local gangrene. Agranulocytosis with angina and stomatitis has been reported. The treatment is immediately discontinued when these symptoms supervene and sodium thiosulfate is given intravenously in gram doses each day for one week. The antidotal effects of B.A.L. or diuretrol are also worthy of trial. Intramuscular injections of bismuth are less toxic than gold therapy and are occasionally beneficial.

Liver extract in doses of 1 to 2 cc given intramuscularly every second day is definitely beneficial.

Wolf and Sulzberger report favorable results following the use of *sulfapyridine* in daily doses of 1 to 4 gm over a period of weeks.

The Sauerbroch Hermannsdorfer salt free diet has been commended by many clinicians on the European continent.

Acute and subacute disseminated lupus erythematosus is treated symptomatically. The sickroom should be kept darkened. Bachr advises administration of *salicylates* to anachorese the fever and arthralgias and to affect favorably the absorption of serous fluid. *Transfusion* of blood from a patient who has recovered from lupus erythematosus or from a donor who has been given gold therapy

is definitely helpful. Whole blood transfusions given early in 250 cc to 500 cc amounts by slow drip (40 drops per minute) may be life-saving since tissues which lack hemoglobin and oxygen repair themselves with difficulty. But plasma for its antibody content may be used in those without anemia. Not more than 500 cc a day should be given if heart failure is suspected or present. Patients with acute or subacute disseminated lupus erythematosus are not given gold therapy. These patients should be put to bed and placed upon a high caloric especially protein diet. Even minor degrees of anemia are significant and should be controlled with liver feeding, liver extract and injection of iron. The diet should be supplemented with vitamin B and C. Lechman advises large doses of mecitric acid (100 mg. two to three times a day).

Repeated small transfusions and intravenous injection of glucose are definitely helpful. Penicillin although usually disappointing should be given a trial in the acute disseminated types. It should be given in very large dosage (60,000 unit every three hours, day and night for fifteen days) because of the possibility of developing penicillin resistant organisms. Penicillin and/or a sulfonamide is indicated in an intercurrent bacterial affection.

TULAREMIA

STAPHYLOCOCCUS *Dere By (see Chad) disease French disease rabbit fever alkali disease*

This is a specific infectious, systemic disease, with or without cutaneous manifestations.

Etiology Tularemia has been observed throughout the United States and in Japan, Russia, and Europe. It is caused by the *Bacterium tularensis*

(*Pasteurella tularensis*). The disease in the human has an incubation period of from one to ten days, with an average of three days. It occurs as a fatal bacteremia in a variety of wild animals, especially rabbits and squirrels, and occasionally in cats and dogs. It may be

nenth patches. Two or three injections are usually sufficient. Third-degree erythema dosage of *ultraviolet light* once a week is also recommended. Five per cent *salicylic acid* in collodion is worthy of trial. The application of solid stick of *carbon dioxide* is occasionally beneficial. *Tuberculin* has been used with benefit in some cases.

Tuberculous Ulcer and Tuberculosis Verrucosa. The local treatment of *tuberculous ulcer* is unsatisfactory. When possible *electrocoagulation* may be helpful. It often relieves pain within twenty-four hours. The lesions of *tuberculosis verrucosa* are treated by *electrocoagulation* or by *curettage* followed by applications of a 10 per cent solution of *zinc chloride*.

Lupus Erythematosus. There is no specific therapy. All possible foci of infection including pulmonary tuberculosis should be excluded. Exposure to sunlight and extremes of heat and cold should be avoided. *Soothing lotions* like 1 per cent Burow's solution and calamine lotion, are employed to relieve acute inflammatory symptoms. Numerous therapeutic measures have been advocated after the acute symptoms of lupus erythematosus have subsided. *Resorcin* and *salicylic acid* are the best topical remedies because they produce local redness and exfoliation within a few days. Topical applications are discontinued for several days after exfoliation when the same lotions are again applied.

The following preparation is suggested by Andrews

Acid salicylic	10.0
Camphor	2.0
Liq. hydrarg. chlor. corr. 1:1000 in alcohol	
III per cent q.s. ad	30.0
Stm. Apply with applicator three times a day for four days.	

Applications of *iodine* pure *phenol* *trichloroacetic acid* and solid *carbon dioxide*

are occasionally beneficial. *Lugol's solution* with 10 per cent acetone applied every other day for several weeks has proved a worthwhile remedy.

The internal administration of *quinine iodoform* *ichthyol* the *salicylates* and *urea chloride* is of value. Cannon advises *tincture of iodine* beginning with 3 drops well diluted in water or milk three times daily and ascending to 10 or more drops three times daily depending upon the patient's tolerance to the drug. The organic *arsenicals* especially *oxyphenarsine hydrochloride* have been of value in some cases. The dose recommended is 0.02 gm in 4 cc of distilled water biweekly and intravenously (Goldberg).

Tonsils and teeth and other possible foci of infection should be investigated.

The chronic forms of this disease are combated successfully by gold therapy bismuth therapy and *sulfapyridine*. Injections of bismuth bismarsan, *tuberculin krysolgan sanocresin* gold sodium thiosulfate and colloidal gold are widely used. Colloidal gold is the most useful gold salt. A 1 per cent solution of gold sodium thiosulfate or colloidal gold in 1 per cent solution of procaine hydrochloride (10 mg gold sodium sulfate or colloidal gold in 1 cc of 1 per cent procaine hydrochloride) is injected intradermally under individual recalcitrant patches. Injections are made at the periphery of the patch and at about three to five points. Results should be appreciable in about ten treatments given once every two weeks. This treatment is occasionally successful when intravenous therapy has failed to give results. It is dangerous to employ gold except in the smallest dose in acute cases and in actively spreading lesions of chronic lupus erythematosus. Gold therapy may in any patient occasionally produce dan-

which is of special interest to the dermatologist.

There is sudden onset of headache, nausea, vomiting, chills, and fever. The temperature falls in a day or two, only to rise and remain elevated for from one to three weeks. This is followed by



Fig. 645 Tularemia. Draining nodular lymphangitis and axillary adenopathy of arm six weeks after onset of tularemic ulcer of the thumb. (Courtesy of Drs. J. M. Hirsch and Dudley C. Smith.)

drenching sweats, weakness, prostration and loss of weight.

In the ulceroglandular type a painful or painless papule, soon becoming a nodule, is found at the site of entry of the organism. This is often on the hands in those who have contracted the disease from handling rodents, and on the lower extremities of those infected by the bite of infected ticks. It is preceded by a day or so, by a slight, painful regional adenitis, which later becomes more marked. The nodule breaks down and liberates a necrotic core exposing an ulcer. The ulcer has soft, ragged raised edges and floor which presents

small granulations from which there is a seropurulent discharge. The ulcer persists for several weeks, and heals spontaneously. The draining lymphatics appear as red streaks, and may be marked here and there by subcutaneous lymphatic nodules (simulating sporotrichosis). In many instances, the regional lymph nodes suppurate as they enlarge, become attached to a reddened (verruca-like) skin, and break through. In other instances, the enlarged nodes remain small, hard and tender sometimes over a period of months. A generalized toxic eruption, which may be macular pap-



Fig. 646 Tularemia. Ulceroglandular form. Note primary nodule on dorsum of hand. (Courtesy of Dr. Ralph Bernstein.)

ular or pustular develops in about 5 per cent of the patients (Francis).

Diagnosis. The ulceroglandular type must be differentiated from *chancere* and *sporotrichosis*. Chancres are not painful as a rule; they show no central necrotic core and their borders are apt to be indurated. In *sporotrichosis*, there are



Fig 643: Tularemia. Generalized maculopapular eruption on fifteenth day of the disease showing many characteristics of erythema multiforme (Courtesy of Drs. J M Hitch and Dudley C. Smith.)

transmitted to humans by contact with or by bites from infected deer flies, wood ticks and other insects, as well as in infected animals. It is not contagious, and transmission from man to man occurs by direct inoculation only. The disease is seen most often in hunters, farmers, cooks, market people, and laboratory workers. Cooking destroys the organism in infected meat.

McCoy and Chapin (1912) described the organism as a small nonmotile, gram negative coccobacillus which grows under aerobic conditions only.

The histological picture is that of a specific granuloma.

Symptoms. Francis, who first found the causative organism in the blood, divides the various manifestations of the disease into the following types: pulmonary typhoid, meningeal, ingested, oculoglandular, glandular and ulceroglandular. It is the ulceroglandular type



Fig 644: Tularemia. Typical primary tularemia ulcers. (Courtesy of Drs. J M Hitch and Dudley C. Smith.)

ULCERS IN SICKLE-CELL ANEMIA

Ulcers on the leg are commonly seen in sickle-cell anemia. The site of predilection is the ankle and lower two thirds of legs. The ulcers may be single multiple, unilateral or bilateral. They



Fig. 647 Ulcer Of leg in sickleemia.

are either nondescript or characterized by sharply margined often punched-out ulcerations, with suppurating base and then vary in size from a quarter to a half dollar or more. The ulcers may appear subsequent to sickle-cell anemia or they may antedate the disease by years.

The mechanism of the development of ulcers in sickleemia is not known, but the

ulcers may be present in the absence of anemia, although sickling is always present. The ulcer may be the only subjective symptom of the disease and the diagnosis can only be established by a blood examination revealing the sickle cells. In active cases, sickling can be found in dried and stained blood smears; in inactive cases, the use of wet preparations, studied for a period of twenty four hours, is required (Diggs and Bibb) for sickling to be completed. The ulcers may closely resemble those due to syphilis, tuberculosis, trauma, and circulatory stasis, but leg ulcers in the adolescent or young Negro should always stimulate a search for sickle cells. Leg ulcers of a similar nature are observed in congenital hemolytic reticula, in which however there is no sickling and splenomegaly.

Sickleemia occurs in Negroes and in those with a strain of Negro blood in them, but the ulcers occur more frequently in Negroes of adolescent age. The sickle-cell trait is often familial and occurs in both sexes. It is believed to be transmitted according to the mendelian law as a dominant characteristic.

Treatment It is the same as for ordinary ulcerations and, when healed, a smooth depigmented scar surrounded by a hyperpigmented areola marks the site of the ulceration. Relapse and recurrence are common and appear to depend upon the grade of the hemolytic anemia for which transfusion may be indicated.

ULERYTHEMA ACNEIFORM

SYNONYMS *Folliculitis ulerythematosus reticulata, atrophoderma aciculata.*

Ulerythema acneiform is characterized by symmetric erythema of the cheeks with follicular comedo-like lesions pre-

senting netlike scarring. The disease begins in early life and tends to form permanent scars before middle life. The

no constitutional symptoms; microscopic and cultural studies for the *Sporotrichum* may be necessary.

Francis bases the diagnosis on (1) a history of having dressed or dissected a wild rabbit or of having been bitten by a deer fly or tick (2) a persistent ulcer preceded by a papulonodule especially on the hand (3) a persistent regional adenopathy and (4) a fever lasting for two or three weeks. Confirmatory laboratory studies consist of (1) isolating the organism from guinea pigs inoculated with the patient's blood or material from the ulcer or the enlarged lymph nodes (2) agglutination of the organism by blood serum obtained after the second week of the illness (3) Foshay's intradermal test, which consists of the intradermal injection of 0.1 cc of nitrated tularemic antigen. The test is read forty-eight hours later. It offers perhaps the best method of early diagnosis.

Prognosis. Recovery is usual, and one attack confers immunity. In severe cases, marked constitutional disturbances persist for months. The mortality rate is from 3 to 5 per cent.

Treatment. Tularemia treatment consists of rest and symptomatic and supportive measures. Only enlarged, softened lymph nodes should be surgically drained.

Foshay's serum given intravenously in doses from 15 to 30 cc ($\frac{1}{2}$ to 1 ounce) on two successive days, and given early is beneficial, but often associated with untoward symptoms and serum sickness in particular.

Streptomycin, according to the National Research Council, is the best therapeutic agent available. It should be given early in 1 to 2-gm (15 to 30 grains) doses daily and intramuscularly (125 to 200 mg in 1 to 2 cc. of isotonic saline solution every three hours) for five to seven days.

the prurigo acute prurigo of Brocq and lichen simplex acutus Vidal.

Giant Urticaria. This form has lesions consisting of circumscribed patches of edema. Lesions occur particularly about the face, lips, ears, eyelids, and cheeks. Burning and itching are marked symptoms. The mucous membrane is occasionally involved. Sudden edema of the larynx may produce grave asphyxia. Giant urticaria is also referred to as angioneurotic edema. (Quincke's disease)

Heredity appears to be an important factor in giant urticaria. The condition is more common among men than among women. Neurasthenia, hysteria, and exophthalmic goiter are occasionally accompanying conditions.

Attacks may appear each month and may coincide with menstruation. The exciting cause remains obscure. It may be emotion, or may result from sudden exposure to cold, or it may be produced by slight trauma.

Nausea, vomiting and abdominal colic may supervene when the mucous membrane of the gastrointestinal tract becomes involved. Attacks are usually afebrile and recurrences are frequent.

Urticaria Haemorrhagica. Hemorrhage rarely occurs. This variety often recurs and is associated with Bright's disease or septicemia.

Pigmented Urticaria or Urticaria Pigmentosa *(cf. French)*. In this form, the urticarial lesion disappears but leave pigmented remains. This pigmented type of urticaria must be differentiated from urticaria pigmentosa (see p. 816).

Urticaria Bullosa. Rarely vesicles and bullae appear in urticaria.

Urticaria Persistens *(cf. Pick)*. This is a form of chronic urticaria characterized by the appearance of small, persistent, wheal-like tumors. These lesions persist for months.

Induced Urticaria. This is the term used for the wheal or welt which develops in most persons with urticaria after the skin is stroked, rubbed or scratched with a dull implement. The whealing as a rule appears rapidly. In induced urticaria the lesions itch in exactly the same manner as the wheals which appear spontaneously in urticaria. As a matter of fact, it may be the only sign of urticaria (subclinical urticaria) with spontaneous wheals altogether absent. Often in subclinical urticaria the patient's only complaint is itching here and there on the body and especially of the palms and soles. The triple response of Lewis is a normal set of reactions which follows cutaneous stimulation and is composed of the following triad:

LOCAL RED REACTION. This is the cutaneous red reaction which follows the lines previously stroked firmly with a blunt instrument. It develops in a few seconds, reaches its maximum within one minute and then fades. It does not depend on the integrity of the skin nerves.

THE FLARE: If the stroking is of sufficient strength or the patient's skin is sufficiently susceptible, the skin to either side of the red line also flushes. It follows local vascular dilatation but does depend on the integrity of the skin nerves.

THE WHEEL. The local red reaction and the flare may be followed by the development of a typical welt. In fact, the skin of certain individuals is especially susceptible to stroking. The whealing produced in these is called "dermographism" (autographism factitious urticaria).

Dermographism is a phenomenon which should be differentiated from induced urticaria, although the resultant lesion is objectively the same. Factitious



Fig 648: Urticaria Reticulata

eruption consists of numerous small areas of atrophy separated by narrow ridges. This produces a reticulated honeycomb or network appearance. Comedones and milia may occur on ridges as well as on depressed areas. The skin has a waxy appearance and is stretched. The affected area is irregularly erythematous. Microscopic examination reveals an atrophic epidermis with loss of papillation. Its course is slow but progressive.

The cause is unknown.

Treatment Thyroid extract in 0.0324 to 0.06 gm ($\frac{1}{4}$ to 1 grain) doses, three times daily over a long period, is beneficial. The scars resulting from this disease are permanent.

URTICARIA

SYNONYMS: *Nettle rash, hives, coldsores.*

Urticaria is an acute or chronic inflammatory condition of the skin characterized by the sudden appearance of white pink, or red wheals surrounded by erythematous halos.

Varieties There are two main types, namely acute and chronic. Almost all urticarias are acute as to the signs and symptoms but vary as to duration. The term "acute urticaria" refers to those which persist several days to several weeks, "chronic urticaria" refers to those which persist for months or years. Varieties of these are the papular giant urticaria, urticaria haemorrhagica, pigmented urticaria, urticaria bullosa, factitious urticaria, or dermatographism, urticaria perstans, and urticaria pigmentosa.

Papular Urticaria This is characterized by discrete and rather small lesions occurring as papules on the extremities and face of infants and young children of either sex. The condition is seen during the first two years of life. It is more

frequent during the summer months. Papular urticaria may disappear with the onset of cold weather only to recur the following summer. Papular urticaria is uncommon among breast fed infants. Its cause is unknown and skin tests for allergy are of no value.

The lesions of papular urticaria possess the same general characteristics of adult urticaria but differ in that they are much smaller and hard and shotlike. As a result of the intense reaction, bullae may form on the surface of the papules. In the early stages they closely simulate insect bites, which are however larger and show more of a tendency to grouping. A central puncture may also be seen under the hand lens. Itching is severe and is often paroxysmal. The condition may persist intermittently over a period of years until the age of nine years, when it usually disappears.

Papular urticaria has been described under *lichen urticatus atrophulus infan*

8. Diseases of the liver and kidney
9. Diseases of the gallbladder
10. Anaphylaxis
11. Parasites infesting the intestinal tract.

Pathology Urticaria is an angioneurosis arising in connection with some vasomotor disturbance. A wheal is an acute localized edema without inflammatory reaction. The upper layer of the cutis, and especially the papillary layer, are very edematous. The tissue does not stain well and appears "blue red" when stained with hematoxylin and eosin.

Symptoms A typical acute urticarial lesion consists of an elevation of the skin with a flat or slightly convex surface pinkish or ivory tinted, and generally surrounded by a reddish inflammatory halo which fades into theough boring skin. It is raised from 1 mm. to 2 mm. and is tense and elastic in consistency. The wheals come out suddenly have a transient existence of a few minutes to several hours, and spontaneously disappear. They may be single or multiple and may occur anywhere on the body. The mucous membranes may be affected as well as the skin. The subjective symptoms of urticaria are sensations of itching, burning, and tingling, which are usually worse at night.

Hypersensitiveness to Cold. Hypersensitiveness to cold is a relatively rare condition, but nevertheless is a definite clinical syndrome. It is characterized by local and sometimes systemic manifestations. After exposure to cold, the local effects on the skin consist of redness, burning, and itching with subsequent swelling and increased local temperature of the part exposed. Actual wheals may appear on the exposed parts. In some patients a part such as the ear or one to several fingers on exposure to cold may become dead white, suggesting an abortive form of Raynaud's disease. This

sensitiveness appears to be vascular rather than cutaneous. After a latent period of from three to six minutes during the development of these local manifestations, a characteristic systemic reaction may develop. However the local manifestations may be the only signs of the person's hypersensitiveness to cold. Systemic reactions usually consist of flushing of the face, a sharp fall in blood pressure, a rise in pulse rate, and in some instances development of syncope. Attacks may be produced by cold wind, cold water, or cold applications, or a sudden change from a warm to a cold environment. After the ingestion of cold water or iced foods, swelling of the lips and, in a few instances, laryngismus may occur. In some patients the reaction is local only whereas in others both local and systemic reactions may occur.

Horton and Brown were able to reproduce the local and systemic reaction by immersion of one hand in water at 48.4 to 50 F (8 to 10 C) for a period of 15 minutes. This provided a test for this syndrome.

TREATMENTS Two procedures were suggested and later found successful. The first procedure was *auto-desensitization*. The patient desensitized himself by daily immersion of the hands in cold water. The temperature of the water was decreased from day to day and the periods of immersion were increased. The second procedure was *passive desensitization* by repeated injection of *histamin* for long periods. Minute doses insufficient to produce a systemic effect were employed, and these were increased gradually. The treatment with histamine was confirmed by Bray in 1932. *Benadryl* gives temporary benefit.

Diagnosis Urticaria is readily recognized by its characteristic lesions, their evanescent nature, their abundance over

urticaria is commonly observed on the back, chest and upper extremities. The welt does not itch and may not appear at once; sometimes 1 to 3 minutes will elapse before it becomes apparent and it may then persist 30 minutes to several hours. It occurs under a wide variety of



Fig. 619 Dermographism

conditions—in the mentally defective in patients with manic-depressive insanity, alcoholism, chronic mercurialism, chronic toxemia, during menstruation in highly emotional individuals and in the apparently normal person.

The passage from physiological to pathological autographism is insensible but a definite wheel occurs in about 5 per cent of apparently normal people. However, a welt can be produced on practically any person with one or more accurately superimposed strokes or with a whip. Actually it is due to the production of an increased permeability of the vessel walls. The triple response as a whole is a set of reactions which can

be produced by various mechanical, physical, and chemical agents and is probably instituted by a varying degree of tissue injury but actually due to the action of a diffusible substance similar to histamine.

Chronic Urticaria. This variety consists in repeated attacks of acute urticaria occurring over a period of months or years.

Incidence. Urticaria may occur at any age and in either sex. The papular variety is for the most part confined to infancy and early childhood. The acute type of urticaria is rare in infancy and old age and more common in females.

Etiology. The causes of urticaria are numerous and varied and result from the action of toxic substances introduced from without or produced within the body in sensitized individuals.

Urticaria may be due to

Exogenous Factors Including

1. Contact with plants and the stings of insects.
2. Hairs of caterpillars, etc.
3. Friction and exposure to extremes of heat and cold and to sunlight (physical allergy).
4. Flannel, wool, and the dyes of clothing.
5. Inhalants (tobacco smoke and perfumes).

Endogenous Factors Which May Be Any of the Following

1. Articles of food, such as fish (particularly shellfish), pork, mangoes, strawberries, etc., mushrooms, some cereals, coffee and chlorinated drinking water at times.
2. Product of decomposition formed in the large intestine.
3. Certain drugs, such as sodium salicylate, aspirin, quinine bromides, iodides, phenolphthalein, luminal, cubeta, copalba, mercury and the sulfonamide drugs.
4. Serum administered for therapeutic purposes.
5. Absorption of toxic products from ruptured hydatid cyst.
6. Toxic products from a focus of infection or during pregnancy.
7. Nervous exhaustion, as a symptom of nervous tension, and as psychoneurosis.

- 11. Diseases of the liver and kidney
- 12. Diseases of the gallbladder
- 13. Anaphylaxis.
- 14. Parasites infesting the intestinal tract.

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Horton and Brown were able to reproduce the local and systemic reactions by immersion of one hand in water at 45.4 to 50 F (8 to 10 C) for a period of six minutes. This provided a test for the syndrome.

TREATMENT Two procedures were suggested and later found successful. The first procedure was *autodesensitization*. The patient desensitized himself by daily immersion of the hands in cold water. The temperature of the water was decreased from day to day, and the period of immersion were increased. The second procedure was *passive desensitization* by repeated injection of histamine for long periods. Minute doses insufficient to produce a systemic effect were employed, and these were increased gradually. The treatment with histamine was confirmed by Bray in 1932. Benadryl gives temporary benefit.

Diagnosis Urticaria is readily recognized by its characteristic lesions, their evanescent nature, their abundance over

covered areas of the body and the accompanying severe itching.

Chronic urticaria bears the same characteristics as acute forms with the exception that the eruptions are less numerous.

The etiologic diagnosis is not usually difficult in acute cases but is often impossible in chronic cases. Skin tests are rarely of diagnostic aid. Each patient in chronic types requires careful study and occasionally the removal of a suspected offender is followed by cure.

Prognosis. Acute urticaria is transitory. It may disappear following treatment or may spontaneously pass away within a few hours or several days. The prognosis of chronic urticaria should be guarded. Recovery depends upon discovery and elimination of the cause.

Treatment. Acute urticaria arising in connection with toxic poisoning is relieved by the ingestion of castor oil and calomel or by an enema followed by colonic irrigation with sodium bicarbonate. The *sympathomimetic drugs* (epinephrine, ephedrine and propadrine hydrochloride) owe their antiallergic action primarily to their vasoconstriction. Subcutaneous injections of 1 to 7 minims of *adrenalin* (1:1000) every half hour for several doses produces relief in most cases. Sedatives like aspirin, the barbiturates, and bromide relieve itching. *Ephedrine sulfate* in doses of 30 mg. (½ grain) by mouth is equally effective. Allergy to the barbiturates and bromides must be borne in mind and discontinued whenever signs of intolerance appear. The opiates are contraindicated.

Saline purgatives and the ingestion of alkalis are beneficial whenever the eruptions are present for some time. *Intestinal antiseptics*, calomel and salol are indicated.

A simple diet with low protein content is recommended. Fish including shell

fish, eggs, meats (except lamb and poultry), cheese, raw fruit (except oranges and grapefruit), berries, tomatoes, pastries, canned foods, hors d'oeuvres, jellies, sweets, cocoa cake, gravies, salad dressing (except lemon or olive oil), alcohol, vinegar, tea and coffee are among the articles of food to be avoided.

Milk, butter, cereals (except oatmeal), poultry, lamb, breadstuffs, ice cream and cooked vegetables (artichoke, beans, beets, broccoli, young carrots, celery, baked cucumber, dandelion, stewed egg plant, greens, lettuce, boiled onion, oyster plant, peas, parsnips, pumpkin, rhubarb, sorrel, spinach, squash, Swiss chard, and vegetable marrow) rarely precipitate urticaria.

Chronic urticaria is difficult to treat. Dietary measures, proper elimination, removal of foci of infection, and the relief of emotional stress are very important in treatment. Intracutaneous tests with various food products are rarely helpful.

Chronic urticaria may be associated with mycotic infection of the skin and with intestinal parasites. Hypersensitivity to carbohydrates is rare. Table salt and acids are occasionally the offending allergens.

Injections of nonspecific proteins like peptone, splenic extract, typhoid, staphylococcus vaccine and streptococcus vaccine are sometimes helpful.

Chronic urticaria has responded in some individuals to the ingestion of an alkaline diet in conjunction with daily doses of 2½ drams (10 grains) sodium bicarbonate.

Cow's milk is the most common of feeding allergen in infants. Infant feeding prepared from soy bean or goat's milk may replace cow's milk. The addition of dilute hydrochloric acid to cow's milk is beneficial. This is prepared by

adding drop by drop 5 cc ($\frac{3}{4}$ dram) of dilute hydrochloric acid to 250 cc ($\frac{1}{2}$ pint) of cold boiled milk containing from 8 to 10 per cent cane sugar.

Ephedrine sulfate is indicated for chronic urticaria unless the blood pressure is high. Injections are given each day to adults in doses of 75 mg ($1\frac{1}{4}$ grams). Children are given three injections each day in doses of 25 mg ($\frac{3}{4}$ grain).

Autohemotherapy diminishes the intensity and frequency of outbreaks. From 2 to 5 cc of the patient's blood is drawn aseptically and immediately reinjected into the buttocks. An injection of 10 cc. is repeated in five days, and subsequently administered once a week for a period of five weeks.

Antihistaminics Much of the clinical and experimental manifestations of anaphylaxis and of allergy are the same despite the fact that both are possible of production by a wide variety of antigens or allergens. Upon this fact has been based the belief that the manifestations themselves are the result of a common denominator which develops after antigen combines at the surface of the sensitized cells and escapes into the blood stream. Dale and Laidlaw (1910) Lewis (1911) and others since have almost certainly proven that one of these substances, in anaphylactic shock at least, less so for allergic states, is histamine or a histamine-like compound and that this agent appears to play a major role. Histamine is a normal constituent of many tissues (Best). Lewis and Grant showed that the whealing of dermographism and of induced urticaria had all the characteristics of histamine induced wheals and Bray showed that the manifestations of cold allergy (increase in gastroacidity, temperature and pulse rate with fall in blood pressure) were those resembling

the action of histamine. According to Lewis, histamine or a histamine-like substance acting directly on the capillary walls is responsible for the skin reaction observed in urticaria. However it does not appear that injections of small doses of histamine are effective in attempts to desensitize to it. Recent studies have been in the direction of substances which will combat it.

In the past several years such histamine antagonists, all chemically related, have been discovered, among these are benadryl hydrochloride and pyribenzamine. The drugs have been used in urticaria (both small and giant), certain types of dermatitis (dermatitis medicamentosa interna, neurodermatitis, contact dermatitis, pruritus vulvae, and some cases of physical allergy) and a variety of other conditions.

The best results—and they are purely symptomatic and the benefits persist but a few hours—have been obtained in the pruritus of subclinical, and in actual acute and chronic urticaria irrespective of the cause. Relief in most cases is obtained only as long as the drug is used. It is an effective palliative which may be curative in some acute cases, but its administration must be continued in most cases indefinitely. The average dose is 50 mg ($\frac{3}{4}$ grain) four times a day. The dosage, however, should be regulated with the results obtained and the toxic effects produced. The drug may be continued indefinitely. In infants, Logan advises 2 mg ($\frac{1}{32}$ grain) per lb of body weight. Children may be given 25 mg ($\frac{3}{8}$ grain) per dose. The side reactions consist chiefly of lassitude, drowsiness, and dizziness in more than half of the patients. Benzedrine, administered at the same time, may overcome this, but the dose may have to be decreased or withdrawn. Dry mouth, nausea, insomnia and bot-

flushes also occur. Desensitization with known allergens must be continued where indicated.

Injections of *extracts of the posterior lobe of the pituitary gland* are helpful in urticaria perstans. In papular urticaria of children a change of residence, possibly to the seashore is often beneficial. In these patients also painting each lesion with undiluted coal tar relieves the itching.

Ellocarpine parathyroid adrenalin dilute hydrochloric acid extract of ergot doryl salt free diet high potassium diet and vitamin therapy have been used with varying results.

Intravenous and subcutaneous injections of insulin are often helpful. The adult dose is 10 units given twice each week. Insulin has been found very helpful in treating cases of chronic urticaria and angioneurotic edema.

Urticaria pigmentosa does not respond to any treatment. Local applications are given merely to relieve itching.

The following dusting powder is very helpful in relieving itching.

Pul camphora	20
Pul zinc o id	20
Amylum	21.0
Sig. Apply freely to relieve itching	

The following antipruritic lotion is beneficial.

Mentholin	0.6
Phenol	0.6
Liq carb deterg	21.0
Pulv tale	30.0
Pulv zinc id	40.0
Glycerini	20.0
Aq camphora	70.0
Spts. vin. rect. (85 per cent)	70.0
Sig. T. relieve itching, shake and apply freely	

A bath consisting of the following preparation is soothing and alleviates itching.

Mentholin	30.0
Spts. vin rect. q.s. ad.	100.0
Sig. Add 1 or 2 teaspoonsful to a hot water bath and bathe for fifteen minutes.	

Temporary relief may frequently be obtained by the application of hot alkali lotions (3 to 5 per cent carbonate or bicarbonate of soda).



Fig. 650: Urticaria Pigmentosa. (Courtesy of Dr. Edward F. Corson.)

Urticaria Pigmentosa

SYNONYM: Xanthelasmodes.

Urticaria pigmentosa is a chronic affection of the skin characterized by pigmented macules and nodules.

Varieties. Three types have been described: a macular type, a nodular type, and a mixed type.

Incidence. The disease usually begins before the sixth month of life. When the lesions commence early in life they are referred to as the "nervus type" and those occurring later in life are referred to as the "adult type." It occurs more often in blonds, and males are more frequently affected than females.

Etiology. The cause is unknown.

The condition has been known to follow measles and vaccination. Cases have been reported occurring soon after birth which suggests a congenital origin.



Fig. 651 Urticaria Pigmentosa. Note the whealing of many of the lesions following friction. (Courtesy of Dr Edward F. Carson.)

Pathology The histology of urticaria pigmentosa is diagnostic. The most characteristic feature is an infiltration of the corium with mast cells. The mast cells are deposited around the blood vessels of the subpapillary layer and upper part of the corium, around the hair follicles and sweat glands. These cells vary in size from small fusiform to large cuboidal. They are most profuse in the nodular type hence its name

"mast-cell tumor." Pigment cells with melanin are present in the basal layer and the layer immediately above it.

Symptoms Seventy per cent of cases appear during the first year of life. The disease occurs in three types: the macular type, the nodular type, and a mixed type.



Fig. 652 Urticaria Pigmentosa. In an adult. Pigmented macules which assume wheal-like fullness when irritated, as by rubbing, are characteristic features. Condition started in early childhood and continued through adolescence to mature womanhood.

The early lesions are urticarial and transient and are replaced either by a brownish macule or a yellowish nodule. The stains which are characteristic of this affection are yellowish or brownish

yellow in color which may assume a violaceous hue on the extremities. They are persistent and may remain for years but finally disappear. The lesions vary in size from a pea to coin sized and larger the macular variety being the largest. The lesions usually remain discrete although in the macular variety they may coalesce and involve large areas.

The back is the site of predilection although other parts of the body including the buccal mucosa are occasionally involved. The lesions when irritated by rubbing become reddened and edema-

tous. Dermographism is usually present.

The subjective symptoms are pruritus; however this is not always present. General lymphadenopathy may be present and is not unlike syphilitic adenopathy.

Diagnosis. It must be differentiated from syphilis, ordinary urticaria, and xanthoma. The color of xanthoma a *chamois leather yellow* is characteristic. The diagnosis of a well marked case of urticaria pigmentosa presents little difficulty. A negative Wassermann reaction and characteristic histologic picture establish the diagnosis beyond all doubt.

Treatment. Treatment is of no avail.

VARICOSE ULCER (SIMPLE LEG ULCER) AND VARICOSE VEINS

The term simple leg ulcer is used to designate those ulcers the basis for whose existence is primarily varicose veins. Trauma appears to be the precipitating factor in most varicose ulcers. The trauma may be from without (a blow or an abrasion due to scratching) or from within in the form of a ruptured vein or a phlebitis. In any case the poor local

Etiology The etiologic factors concerned in the production of varicose veins are apparently multiple. In some, as Delbet holds, mechanical pressure in the abdomen causes the retrograde incompetence of the venous valves by dilating the veins. This process begins with the femoral vein and continues down the superficial saphenous system. This theory



Fig. 653 Varicose Ulcer. Small, acute. Typical location on lower lower third of leg.

nutrition due to the varicose veins and the temporary devitalization predisposes to the prolongation of the injury and to infection by the common, cutaneous, pyogenic organisms—staphylococci and streptococci in particular. It is probable also that in the presence of varicose veins, with the associated cutaneous nutritional difficulties, a primary infection with these organisms (impetigo eczema) occasionally originate the simple leg ulcer. According to Embourand, chronic simple leg ulcer is in the nature of a chronic streptococcal chancre.

would easily explain the varicose veins of pregnancy and of intraabdominal origin especially pelvic tumors, and varicocities seen in laborers. The theory of degeneration and resultant insufficiency of venous valves proposed by Klotz explains many cases of varicose veins. There is some reason to believe that certain specific infectious diseases, such as typhoid fever, scarlet fever, pneumonia and influenza may cause degenerative changes in the veins leading to the development of varicocities.

There are some associated factors

which are so frequently observed that they may play an important predisposing role in preparing the way for varicose ulcers. Occupations that require long hours of standing or those that require the lifting of heavy objects tend to increase the pressure of the blood in the venous circulation. A similar result is



Fig. 654: Ulcer Cruri and Varices.

produced by any chronic pulmonary affection that causes constant coughing or pressure. Hereditary predisposition is likewise believed to play a role.

Most cases of varicose veins develop before the fourth decade of life and this disease is approximately four times as prevalent in females as in males.

Symptoms. Simple leg ulcers are acute or chronic—using the term “acute” to indicate an ulcer of one week to several months duration under these circumstances furthermore the ulcer itself is an acute process. Acute ulcers often develop as recurrences in the scars of former ulcers. The acute varicose ulcer is single sometimes multiple pinhead to dime-sized and painful but especially tender. It has a soft base soft borders, and a

punched-out, more or less rounded, oval or polycyclic appearance. The borders are frequently undermined. The floor after cleansing is found to be hemorrhagic and covered with fine granulations which bleed easily. The serous discharge is apt to be blood tinged. However when crusts form, pus is usually found beneath them. The skin surrounding the ulcer is commonly pigmented and not infrequently reddened.

Although varicose ulcers may occur on both legs, they are generally unilateral. The site of a varicose ulcer is diagnostic. It is apt to be located on the lower third, inner side of the leg usually above the malleolus. A varicose ulcer tends to persist but whether it does so by itself or as a result of the coalescence of several ulcers, many of the above characteristics are gradually lost. As they enter into the chronic stage they become less painful and less tender. The discharge assumes a purulent character the borders become firm and scleroid. They may increase in depth as well as in extent and scleroedematous changes occur to a varying degree above and below and sometimes for a considerable distance around, the ulcer whose size may now be sufficient to involve a large part of the leg and even its entire circumference. Repeated mild to moderate attacks of lymphangitis may eventually lead to a pseudoelephantiasis stage of varying degree (see p. 839). In other instances, a contracting, sclerotic change in the subcutaneous tissue results in complete obliteration of the subcutaneous fat and atrophy of the skin with band like stricture of the lower third of the leg. Darier has pointed out that this *dermatosclerosis* may develop even before ulceration. The term “ulcus hypostaticum” has been used for those ulcers where varicose veins cannot be demonstrated.

Complications These occur either with varicose veins or in a varicose ulcer. In addition to ulceration, the following may complicate varicose veins: subjectively a heavy sensation in the legs and pruritus; objectively an eczema that is dry (eczema squamosum) or moist, eroded, and crusted (nummular eczema, eczema rubrum, and infectious eczema, toxic dermatitis); pigmentation of the skin of the lower part of the leg, the result of repeated small intracutaneous hemorrhages, areas of lichenification even, edema of the legs, and in occasional instances a progressive sclerodematous state allied to elephantiasis nostras. "Dermatitis hemostatica" or "stasis dermatitis" is the term used to designate the bluish red, sometimes edematous, often pigmented, cutaneous state which develops on the legs and ankles of patient with long standing varicosities. Itching is often present. The term has at times been used synonymously with varicose eczema. There are at least two etiological types of varicose eczema. One of which is the most frequent, there are varicose ulcerated patches, up to involvement of half or more of the skin of the leg and ankle, of thickened reddened skin with eroded and crust-covered areas, all the result of rubbing and scratching on a pruritic skin. The pruritus is the result of vitiated cutaneous circulation due to the venous insufficiency. In the second type, lesions, also of varying extent, differ in no way from those observed in infectious eczematoid dermatitis or nummular eczema. It is probable that these lesions are really bacterial, also originally developed on a pruritic base. Both types may be present at once and the same time.

Although varicose ulcer is a frequent complication of varicose veins, probably the most frequent complication is thrombophlebitis, most commonly the following

in the superficial circulation, particularly the great saphenous vein in the leg. The thrombophlebitis may be of a recurrent or migrating type. In this connection it is well to remember that thrombophlebitis is also seen following operations, mechanical and chemical injuries, acute infections, and after childbirth. The condition itself is characterized by the sudden development of small pain and slightly larger-sized, tender discrete single or multiple nodules, especially on the lower extremities. They occur in crops, along the venous channels, and persist two to three weeks when they tend to resolve completely. From the standpoint of economics, ulcer is probably the most serious of all of the complications. The great chronicity of this disease with its usual tendency to recur makes it a problem of prime importance. Unfortunately each attack of varicose ulcer further destroys the structure of the overlying skin so that, after a period of years, the affected tissues may be said to be in a state of potential necrosis, developing ulcers often without cause, long after the varicosities which caused the original ulcer had been destroyed.

Diagnosis Varicose Veins The diagnosis of varicose vein is a matter of simple observation with the patient in the erect posture the dilated veins become prominent and on palpation the tension in the veins is found increased. However in order to treat varicosities intelligently together with their complications, it is important that the examiner determine the condition of the venous circulation, i.e. whether he is dealing with a simple dilatation of veins, or whether there is a reversal of the normal direction of venous blood back through the saphenous valve and any of the communications between the superficial and deep venous systems. The Trendelenburg test is a procedure

which are so frequently observed that they may play an important predisposing role in preparing the way for varicosities. Occupations that require long hours of standing or those that require the lifting of heavy objects tend to increase the pressure of the blood in the venous circulation. A similar result is



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is carefully searched for and the communication is cut between ligatures.

Varicose Ulcer. The diagnosis of an acute varicose ulcer is generally not difficult, chronic varicose ulcers require differentiation from ulcers due to the following, although any of them may develop in patients who also have varicose veins: arteriosclerosis, thromboangiitis obliterans, diabetes mellitus, primary anemia (sickle cell and pernicious), secondary anemia, infections such as syphilis, tuberculosis and blastomycosis, drugs (bromides especially), ulcerative type of Bazin's disease, and cancer. A diagnosis of chronic varicose ulcer is not permissible until these conditions, syphilis in particular, have been excluded or their influence in the presence of varicose veins and an atypical ulcer determined. Clinical and serological studies for evidences of syphilis are indicated in all chronic leg ulcers.

Treatment. This consists in treating the varicose veins as well as the ulcer. In many instances, obliteration of the veins surgically or by sclerosing injections will lead to disappearance of complications (ulcers, eczema, etc.)

Varicose Veins. In cases where there is a definite backflow through either the saphenous-femoral junction or through a communicating vein in the lower thigh or leg, ligation of the incompetent veins should be done first. Failure to observe this fundamental fact is the chief reason for the discredit which befell the so-called injection treatment of varicose veins. Chemical thromboses which are subjected to a high venous pressure are soon canalized, and collateral veins which were previously normal in calibre are distended to such a point that in a few weeks or months the varicosities may be as bad or worse than they were prior to treatment.

Before considering the occlusion of veins by chemical agents, it must be established that the venous blood is moving through its proper channels, i.e. toward the heart and from the superficial circulation to the deep venous circulation.

The presence of superficial thrombophlebitis is no contraindication to venous ligation, if the venous system is incompetent. In fact this operation if it includes high saphenous ligation makes the possibility of pulmonary embolism much less likely and in addition lessens the period of disability. The use of *sympathetic block with procaine* is excellent for the relief of the severe pain which may accompany this condition. The practice of enforcing prolonged periods of bed rest, wet pads, and elevation in thrombophlebitis accomplishes no useful purpose.

In cases where operation fails, the cause of failure is practically always due to communication between the deep and superficial systems which was missed. A second operation, usually under local anesthesia would remedy the condition.

Sclerosing agents with the object of producing permanent thrombosis, are only indicated in cases of varicose veins with the Trendelenburg test nil. In selecting a sclerosing agent it is wise to consider possible toxicity and idiosyncrasy. It may be said that the more efficient agents are more likely to cause unpleasant reactions. *Sodium morrhuate* 5 per cent, from 2 to 5 cc., injected directly into the vein, is widely used and is quite effective. A safer preparation, at least from the standpoint of reaction and in the same dosage, is a mixture of solutions of 25 per cent glucose with 12½ per cent sodium chloride. The cramps produced by this material may be intense but they are usually gone by the time the patient is ready to leave. Persistent edema may

designed to prove the presence or absence of a reverse flow. A Trendelenburg positive is a reversal in the flow of venous blood through the valve at the saphenofemoral junction or any communication between the superficial and deep circulation in the thigh.

A Trendelenburg negative is a reversal of the flow of blood through any of the communicating veins in the leg. A Trendelenburg double is a combination of both conditions as described above.

A Trendelenburg nil indicates that there is no reversal in the flow of blood between the deep and superficial systems, the varices being due to simple dilatation of the superficial veins.

In order to perform the Trendelenburg test have the patient stand up exposed to the groin. The veins will fill when the veins are clearly visible mark their course with mercurochrome. This will mark their position at all times during the test and will be useful when the veins are collapsed.

Have the patient lie prone on the examining table, raise the leg and allow the blood to drain from the varicosities. Take a hand towel which has been folded to make a pad about 4 inches square and press it firmly over the saphenofemoral junction in the thigh. Now the patient is instructed to stand up. If the veins fill slowly (longer than 30 seconds) and do not become as tense as they were prior to the application of pressure in the groin the main reversal of blood flow is through the saphenous valve and ligation of this vein and its communications will remedy the condition. This may be proven by releasing the pressure in the groin the varicosities will fill up almost instantly.

If pressure over the saphenofemoral junction has no effect on the filling of the varicosities, the communications between

the venous systems is lower down in the thigh or may be entirely in the leg. Each suspected area of communication must be tested separately for its effect on the varicosities below its level and wherever an incompetent communication is found it should be marked for ligation.

A simple method of checking the sites of reversal of blood flow through communicating veins is to have the patient stand and let the veins fill. With one finger empty the blood from the varicosities by gentle pressure over the vein pushing the blood upward and hold this pressure over the site of a suspected incompetent communication. If this is truly an incompetent communication the varicosities distal to it will remain empty or under low tension.

Before ligation or occlusion by injection of a sclerosing agent the possibility of a compensatory dilatation of the superficial venous system to overcome the blocking of the circulation by deep thromboses must be excluded. It is obvious that obstruction of both systems (superficial and deep) would not contribute to the well being of the patient.

To test the patency of the deep circulation several tests have been devised. One of the most reliable is Perthes modification of the Trendelenburg test. A venous tourniquet is applied above the knee and the patient is instructed to walk briskly for five minutes or more. If the veins empty on exercise and the patient tolerates the tourniquet, it is certain that the deep venous system is functioning and the patient is suitable for whatever type of treatment is indicated.

Actually in preparing a patient for treatment each incompetent valve is sought out and marked with 1 per cent gentian violet in alcohol or a 20 per cent solution of silver nitrate. At operation each communication previously marked

is carefully searched for and the communication is cut between ligatures.

Varicose Ulcer. The diagnosis of an acute varicose ulcer is generally not difficult, chronic varicose ulcers require differentiation from ulcers due to the following although any of them may develop in patients who also have varicose veins: arteriosclerosis, thromboangiitis obliterans, diabetes mellitus, primary anemia (sickle cell and pernicious), secondary anemia, infections such as syphilis, tuberculosis and blastomycosis, drugs (bromides especially), ulcerative type of Bazin's disease, and cancer. A diagnosis of chronic varicose ulcer is not permissible until these conditions, syphilis in particular, have been excluded or their influence in the presence of varicose veins and an typical ulcer determined. Clinical and serological studies for evidences of syphilis are indicated in all chronic leg ulcers.

Treatment. This consists in treating the varicose veins as well as the ulcer. In many instances, obliteration of the veins surgically or by sclerosing injections will lead to disappearance of complications (ulcers, eczema, etc.)

Varicose Veins. In cases where there is a definite backflow through either the saphenous-femoral junction or through a communicating vein in the lower thigh or leg, ligation of the incompetent veins should be done first. Failure to observe this fundamental fact is the chief reason for the disaster which befell the so-called "injection treatment of varicose veins." Chemical thromboes which are subjected to a high venous pressure are soon canalized, and collateral veins which were previously normal in calibre are distended to such a point that in a few weeks or months the varicosities may be as bad or worse than they were prior to treatment.

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follow the indiscriminate use of sclerosing solutions.

Varicose Ulcer **PROPHYLAXIS** Most varicose ulcers and other complications of varicose veins can be prevented by the constant use of elastic stockings or the daily proper application of elastic bandages (B & B tensor) to include the foot and leg to the knee. Such bandages are removed at bedtime after the patient is in bed and reapplied in the morning before stepping out of bed. The constant use of such a support will usually prevent ulcer recurrence, but venous ligation may be necessary. Standing in one position for any length of time is best avoided.

CURE It is impossible to cure varicose ulcers without improving the local venous circulation or in the presence of edema. Rest with the affected limb elevated may be necessary. However, acute ulcers and many small chronic varicose ulcers can be cured without recourse to bed rest. One of the best ambulatory methods is the so-called rubber sponge or venous heart method described by McPheeters. The procedure is as follows:

- (1) Place petrolatum gauze next to the ulcer which has been painted with 3 per cent aqueous solution of gentian violet or with suspension of sulfathiazole (20 per cent) in 1:1000 phenetrol. Maceration of contiguous skin from spreading of occluded edema can be avoided by protection with application of Levasor paste.
- (2) Place several fl. oz. of gauze for drainage over this petrolatum gauze and then add several layers of sheet wadding to protect the underlying skin from being irritated by the rubber sponge.
- (3) The rubber sponge is now applied to cover the ulcer completely and if much edema is present to encase the entire leg. Hold the sponge in place with a gauze bandage. In the presence of edema, paint the skin with compound tincture benzoin and apply a longitudinal strip of adhesive plaster on the sides of the leg. It will keep the bandage from slipping and prevent the excessive growing of edematous tissue.

- (4) Apply an Ace bandage to include the foot and leg to point below the knee.
- (5) I direct the patient to walk as much as possible.
- (6) Do not re-dress for seven to ten days if possible.

Another ambulatory method commonly used in recent years has been the application of the zinc oxide-glycerogelatin boot of Unna of which there are a number of modifications. This is applied after a twenty-four to forty-eight hour period of bed rest designed to reduce as much of the edema as possible. The surface of the ulcer is then cleansed of debris and painted with a 3 per cent aqueous solution of gentian violet or a 20 per cent suspension of sulfathiazole. In the presence of eczema the following have been found beneficial:

I	
Pul. Zinc Oxide	8.0
Petrolatum	32.0
Phenol	0.6
C'lonel	1.0
M. Applr. t. eczematous reac.	
II	
Pul. Zinc Oxide	8.0
Petrolatum	32.0
Ithammol	1.6
III	
Nastala	

Unna's paste is heated gently in a water bath or double boiler until it becomes fluid in consistency. It is then applied with a soft paint brush to the foot and leg and quickly covered with a simple spiral bandage. Then another layer of the paste until four layers of paste and three of bandages have been applied. A light gauze dressing is applied over the entire bandage. Re-dressings are made when secretions show through the boot or at seven to ten-day intervals. Ready for use zinc-oxide-gelatin impregnated bandages (gelocast—Duke Laboratories) are widely used.

In some especially chronic ulcers complete bed rest may be necessary. Under these circumstances, inflammation

and edema tend to disappear under the additional influence of constant, wet, (4 per cent) boric acid or isotonic salt solution compresses with the leg kept continuously slightly elevated. Infection is still further reduced by daily applications of the aqueous solution of gentian violet powdered sulfathiazole or a 20 per cent suspension of this drug. As the infection disappears and granulations develop, cure may be hastened by means of small skin grafts removed from the thigh. Excision of a chronic ulcer may occasionally be necessary.

In conjunction with absolute rest the following slightly modified formula of Murray and Shear's blood paste has been used with success in chronic leg ulcers. Into a 500-cc. flask or bottle, by means of a vacuum, aspirate 450 cc. of a type O, red-cell concentrate remaining after plasma is removed. Powder 2.5 gm. (37.5 grains) of actocin on to the surface of 75 cc. of a 1:1000 solution of phenmerol placed in a sterile pyrex glass beaker. Cover and allow it to stand until gelatinous (three to four hours). Sterilize this

for twenty minutes at 212° F (100° C) in a water bath. Allow to cool. With a sterile spatula, take 40 gm. (600 grains) and add it to the 450 cc. of red-cell concentrate. Thoroughly agitate. Store at 32 to 41° F (3 to 5° C). Cleanse the ulcer with isotonic saline solution and dry gauze. Apply paste to ulcer with sterile cotton applicator once daily. If the ulcer is located in tissues having a cartilaginous induration, its borders must be freed by circular or numerous parallel incisions to enable new capillary and granulation tissue formation.

Ulcers sometimes persist after the venous circulation has been improved. Tyrothricin locally and occasionally penicillin injection or oral sulfonamides are indicated. On occasion, the obliteration by sclerosing injections of all feeder veins near an ulcer will markedly improve the lesion. The management of this type of ulcer is frequently very trying. Careful attention to detail and patience together with ingenuity are rewarded frequently enough to warrant the extra effort.

VERRUCAE (WARTS)

Verrucae are benign circumscribed epithelial tumors.

Etiology. Verrucae are of infectious origin, the exact nature of which is unknown. Kingery obtained a filtrable virus from lesions and demonstrated that inoculation is followed within from four to six weeks by the appearance of warts. Warts are, therefore, infectious and autoinoculable. They have occurred about sites of scratch marks, abrasions, and on contiguous surfaces. Warts are species specific and although certain animals (dog, cattle, rabbit) can acquire warts, they do not transmit to man. It appears that immunity to warts may oc-

cure in human beings. Findlay found that, after inoculating himself with three crops of warts, he became immune to further inoculations. It has been stated that immunity normally occurs in about three years.

Pathology. The pathology of verrucae is that of a hypertrophy involving the horny epithelial cells and the papillae which show definite dilated capillary loops. Acanthosis is chiefly present in the prickle-cell layer of flat warts.

Varieties. Four types of warts are recognized.

Verruca Vulgaris. This is the common wart among children. It occurs on the

follow the indiscriminate use of sclerosing solutions

Varicose Ulcer **PROPHYLAXIS** Most varicose ulcers and other complications of varicose veins can be prevented by the constant use of *elastic stockings* or the daily proper application of *elastic bandages* (B & B tensor) to include the foot and leg to the knee. Such bandages are removed at bedtime after the patient is in bed and reapplied in the morning before stepping out of bed. The constant use of such a support will usually prevent ulcer recurrences but venous ligation may be necessary. Standing in one position for any length of time is best avoided.

CURE It is impossible to cure varicose ulcers without improving the local venous circulation or in the presence of edema. *Rest* with the affected limb elevated may be necessary. However acute ulcers and many small chronic varicose ulcers can be cured without recourse to bed rest. One of the best ambulatory methods is the so-called *rubber sponge or venous heart* method described by McPheeters. The procedure is as follows:

- (1) Place petrolatum gauze next to the ulcer which has been painted with 3 per cent aqueous solution of gentian violet or with a suspension of sulfathiazole (20 per cent) in 1:1000 phenol. Maceration of the skin from spreading of occluded exudate can be avoided by protective application of Lassar paste.
- (2) Place several strips of gauze for drainage over the petrolatum gauze and then add several layers of sheet wadding to protect the underlying skin from being irritated by the rubber sponge.
- (3) The rubber sponge is now applied to cover the ulcer completely and, if much edema is present to encase the entire leg. Hold the sponge in place with gauze bandage. In the presence of edema, paint the skin with compound tincture benzoin and apply longitudinal strip of adhesive plaster on the sides of the leg. It will keep the bandage from slipping and prevent the excessive growing of edematous tissue.

- (4) Apply an Ace bandage to include the foot and leg to a point below the knee.
- (5) Instruct the patient to walk as much as possible.
- (6) Do not re-dress for seven to ten days if possible.

Another ambulatory method commonly used in recent years has been the application of the *zinc oxide-glycerogelatin boot of Unna* of which there are a number of modifications. This is applied after a twenty-four to forty-eight hour period of bed rest designed to reduce as much of the edema as possible. The surface of the ulcer is then cleansed of debris and painted with a 3 per cent aqueous solution of *gentian violet* or a 20 per cent suspension of *sulfathiazole*. In the presence of eczema the following have been found beneficial:

I	
Ful. Zinc Oxide	2.0
Petrolatum	50.0
Phenol	0.5
Calomel	1.0
M. Appl. to eczematous areas.	
II	
Ful. Zinc Oxide	2.0
Petrolatum	51.0
Ichthammol	1.6

Naftalin

Unna's paste is heated gently in a water bath or double boiler until it becomes fluid in consistency. It is then applied with a soft paint brush to the foot and leg and quickly covered with a simple spiral bandage. Then another layer of the paste until four layers of paste and three of bandages have been applied. A light gauze dressing is applied over the entire bandage. Re-dressings are made when secretions show through the boot or at seven to ten-day intervals. Ready for use zinc-oxide-gelatin, impregnated bandages (gelocast—Duke Laboratories) are widely used.

In some especially chronic ulcers complete bed rest may be necessary. Under these circumstances, inflammation

cult. At times, verrucae must be differentiated from *pidemodysplasia verruciformis* (Lewandowsky-Lutz) which tends to appear shortly after birth and before puberty. This latter is a genodermatosis in which the eruption according to Wise, is polymorphous. Most lesions

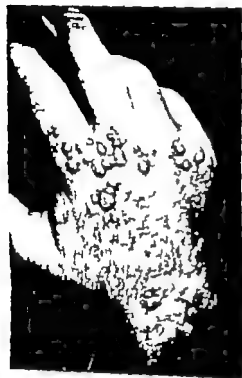


Fig. 657 Verrucae V. Igaria. (Courtesy of Dr. C. C. Thomas.)

are verrucous, and often resemble juvenile flat warts, especially on the face. Others are flat topped, lichenified, rounded or polygonal, pink to brown papules. Sites of predilection are the face, neck, and extremities, especially the dorsa of hands and feet. The lesions may show thin, easily detached, grayish white friable scales. There is a tendency to confluence, the production of elevated lichenified plaques, and occasional epitheliomatous changes in one or several lesions.

Goldsman's transparency test is useful in the differential diagnosis of a planar wart from callus and corns. It consists in covering the suspected lesion with xylene or oil and examining the result under a strong light. The papillomatous projections or areas of acanthosis of a wart appear as discrete circular plugs.

Treatment. There is no uniform successful therapy although there are many methods. Many disappear spontaneously. Satisfactory results have been produced in verruca plantaris by the internal administration of 250 cc (1 pint) of lime-water each day; calcium lactate 0.5 gm. (8 grains) three times each day before meals, small doses of



Fig. 658 Verrucae

Fowler's solution 0.008 cc (1 minim) three times each day; protoiodide of mercury in doses of 0.01 gm. ($\frac{1}{4}$ grain) three times each day; magnesium sulfate 0.6 gm. (10 grains) three times each day; dilute hydrochloric acid, 1 cc. (15 drops) three times each day; and the interdigital injection of bismuth.

Verrucae (Warts)

fingers and backs of the hands, and other parts of the body surface.

Verruca Juvenilis This is seen chiefly on the forehead, neck, dorsum of the hands, wrists, and knees. These warts



Fig. 655: Multiple Warts.

appear in small or large numbers, vary in size from a pinhead to a small pea, and are frequently associated with *verruca vulgaris*. They are flat and but slightly elevated above the skin surface.

Verruca Plantaris It appears on the ball of the foot and heel and assumes the appearance of a corn from the firm horny ring surrounding it. *Verrucae plantaris* are often bilateral in incidence and symmetrical in appearance. Tenderness makes walking painful. They may be single or multiple and in these are apt to be well developed. Occasionally they are dry, painless, spread over large areas, very superficial and patchlike (mosaic type of Montgomery). The mosaic wart is limited almost invariably to the sole and at pressure points. It has a

diffuse, not sharply margined border with a rough granular surface, which when pared shows closely packed, soft corn like segments. This type is very resistant to therapy.

Verruca Digitata Acuminata or *Filiformis* These appear on the genital and perianal regions, and may be seen about the mouth, on the scalp, on the eyelid and nostril. The lesion is a minute thread like flexible wart like newgrowth consisting of a filamentous process rarely exceeding $\frac{1}{8}$ inch in extent and having a base measuring 1 mm in diameter. Digitate lesions present several finger like projections. *Verrucae filiformis* may occur in large numbers and assume a cauliflower



Fig. 656 Papilloma. Of right breast.

appearance. In the genital area they are often termed "condylomata acuminata" or "venereal warts." They frequently occur in conjunction with gonorrhea, when they are known as "gonorrheal warts." *Verrucae filiformis* are seen on the vulva and become associated with leukorrhea.

Diagnosis This is generally not diffi-

The method of treatment is determined by the patient's age, the type of wart and its location. Radium x-ray and fulguration give the best results. Verrucae juvenilis is also treated by washing the involved areas several times each day in Vlemmick's solution or in an aqueous solution of sodium carbonate. 50 gm. (1 ounce) of sodium carbonate

of 60 per cent salicylic acid paste as follows.

A moleskin plaster having a hole the size and shape of the patch is affixed to the affected area. The paste with a thickness of $\frac{1}{32}$ inch is spread within the holes and retained in place with adhesive plaster. The application is repeated every five to seven days. The



Fig. 660 Verrucae Warts (condiomyoma acuminata)

t 250 cc (1 pint) of water. Roentgenotherapy is advised whenever this treatment is unsuccessful. Verrucae vulgares and plantaris are the most susceptible to x-ray therapy. For large and plantar warts, the preferable dosage is an initial 900 r then 600 r repeated twice ten days apart, with the lesion well shielded with lead.

The author has obtained favorable results by the use of the following preparation:

Paraffin (Verech)	0.5
Colodion	2.0

Use: Apply with applicator every other day for a period of three weeks.

For the planter mosaic wart, Montgomery advises the repeated application

macerated tissue is cut away at each visit and a new shield applied. Finally when the wart is exposed it is shielded with strong silver nitrate solution (1 grain to 1 minim). Several such dressings are made each time after paring down the black eschar. The method is somewhat painful but not unendurable. Recurrences are prevented by rubbing in a 30 per cent salicylic acid ointment nightly using a finger cot.

For *condiomyoma acuminata* (1) Anderson suggests 10 per cent *trypodophyllin resin* (U.S.I.) applied with a camel hair brush to cotton swab. The surrounding normal tissue should be surrounded with *Lassar* paste.

Biberstein obtained 75 per cent success in treating 130 cases of warts by immunotherapy. He excised the warts and ground them in mortar with normal salt. The mixture is allowed to stand for twenty-four hours and then passed through a Berkefeld filter. It is then

The injection into each wart of a few drops of soluble bismuth or a few drops of arspenamine in isotonic salt solution leads to their spontaneous disappearance.

Autosuggestion is often followed by the disappearance of warts whether single or multiple. Although suggestion may be



FIG. 659. Verrucae Plantarum. Left: Mosaic wart before application of keratolytic agent. Right: The typical mosaic formation revealed on paring after an application of 60 per cent salicylic acid paste. (Courtesy of Dr. A. H. Montgomery.)

heated to 50° C. and sufficient phenol added to make a 0.5 per cent solution. Subcutaneous injections of this solution are given twice each week in doses ranging from 0.2 to 0.4 cc. Injections of a few drops of an aqueous solution of urea repeated twice a week have proved valuable.

The better known local remedies are excision, fulguration, solid carbon dioxide, trichloroacetic acid, chromic acid, caustic potash, iodine, silver nitrate, phenol, and equal parts of phenol and oil of thuya.

used alone, it is best used in conjunction with any of the methods herein noted because of the frequency with which warts return following their destruction with physical or chemical agents. Suggestion should always be tried in children with many warts. Many ways of suggestion have been used. Sheer treated fifty children suffering with verrucae. They were led blindfolded one by one into an adjoining room and the verrucae were painted with eosin using firm pressure. One month later 40 per cent of the children were free of warts.

only to failure to ingest an adequate supply for the body needs, but also to failure to convert the dietary source to the utilizable form this is met in biliary and liver disturbances.

It is well to remember too that mineral oil exerts a selective solubility for carotene which does not exist for natural vitamin A. Therefore, mineral oil taken in proximity to mealtime may delete practically all of the dietary source of this vitamin. When mineral oil is used, it is well to fortify the diet with some natural source of vitamin A. In fact, proprietary preparations are now available which combine therapeutic doses of mineral oil with the day's requirement of the natural vitamin. These products should be used when mineral oil is indicated.

It should also be remembered that the body will store vitamin A and the other fat-soluble vitamins; it will not store the water-soluble vitamins. When the metabolism or utilization of the essential food elements is abnormal, symptoms of vitamin deficiency of the water-soluble group are likely to occur first. This does not mean however that symptoms of the fat-soluble vitamin deficiency will not follow. In fact, depletion of this mine may produce symptoms of fat-soluble vitamin deficiencies more quickly than otherwise.

In chronic diseases, such as chronic ulcerative colitis, diarrhea, liver and biliary disorders, diabetes, peptic ulcer, prolonged catharsis, obstructive lesions of the gastrointestinal tract, etc. symptoms of avitaminosis more commonly occur.

Pathology In avitaminosis A, the skin is dry and scaly and later becomes the seat of a papular eruption. These papules consist of keratotic plugs in the hair follicles. After these keratotic plugs have disappeared the skin is hairless and

the sites of the follicles are craterlike in appearance. Atrophy of the sudoriferous glands and a keratinizing metaplasia of their ducts prevent sweating. The sebaceous glands also undergo keratinizing metaplasia and finally atrophy. The sites of predilection of keratotic folliculitis in order of sequence are the antero-lateral aspect of the thighs, and the posterior lateral aspect of the upper part of the forearm, extensor surface of the arms and the shoulders, and less commonly the abdomen, back, buttocks, and finally the forepart and the back part of the neck.

The pigmentation of the skin and conjunctiva is increased while that adjoining areas of skin proximal to the hyperkeratotic hair follicle is decreased. According to Otto A. Bessey and S. D. Wolfback, the involvement of the eye occurs late in human beings.

The earliest demonstrable changes are metaplasia of the epithelium of the conjunctiva (xerosis conjunctiva) metaplasia of the cornea (xerosis cornea) and Bitot's spots. Bitot's spots are areas of conjunctival thickening occurring near the limbus of the eye and have the appearance of dried foam. Atrophy and metaplasia of the lacrimal glands and ducts are also encountered. The cornea may become vascularized, edematous, and the seat of leukocytic infiltration.

Symptoms The first symptoms of avitaminosis A are a feeling of discomfort, minor aches and pains, a condition generally known as "ill health." The characteristic symptoms include changes in the adaptation of the eye to light intensity leading eventually to night blindness, retardation of growth in the young, and changes in type of epithelial lining of the respiratory tract, genitourinary tract, alimentary tract, eye and paracutaneous glands, salivary glands, sweat

(2) Haiber suggests the use of *podophyllin* (5 per cent) in *tannic acid*

(3) MacGregor has obtained excellent results with a 25 per cent suspension of *podophyllin resin* (B.P. in liquid paraffin B.P.) The suspension is thoroughly shaken and applied freely to affected area

and crevices between the lesions. A protective dressing is needed. Hard crusts from shriveled warts develop within a few days and should be removed and a second and third application made if necessary. Ordinary warts are not affected by this therapy.

VITAMINS IN SKIN DISORDERS

Haphazard vitamin therapy in the dermatoses is as disappointing as similar therapy is for many systemic diseases.

Specific results with vitamins have been obtained in ariboflavinosis (or cheilosis), pellagra, phrynoderma in some cases of pityriasis rubra pilaris and keratosis follicularis, and in certain seborrheids.

Favorable results have been obtained by some investigators in a wide variety of other conditions among which may be noted moniliasis, rosacea, xerosis, senile vaginitis, keratosis pilaris, acrodermia and herpes zoster. Vitamins have been used as a preventive for arsenphenamine dermatitis.

Abnormalities not related to deficiency of one single vitamin are seen in malnourished persons such as dry brittle hair, facial seborrhea and a patchy facial pigmentation.

Vitamins are complex organic substances, minute quantities of which produce specific biological effects, the lack of which leads to an impaired cellular metabolism. A vitamin deficiency produces certain clinical symptoms which are described under Avitaminosis.

Varieties. The known vitamins may be classified in two general groups, fat soluble and water soluble. Fat-soluble vitamins of clinical importance are vitamins A, D, E and K. The members of the B complex and vitamin C belong to the water-soluble group.

A deficiency or absence of any of these vitamins produces definite clinical symptoms and is a variety of an avitaminosis.

Avitaminosis A

This is a nutritional abnormality produced either by a lack of vitamin A in the daily diet or by failure to utilize or absorb that which is consumed.

Incidence. Recent surveys show that vitamin A deficiency is of more frequent occurrence than heretofore recognized. Determinations of the vitamin A level in the blood and discovery of the existence of night blindness through the use of a biophotometer are responsible for the recognition of the earlier stages of this deficiency disease. In a series of cases taken at random from a children's clinic, one author (Elmer O. Dahl of Minneapolis) reports a 21 per cent deficiency in vitamin A while Jenas in Iowa City found a 10 per cent deficiency in vitamin A and 5 per cent on the borderline.

Etiology. In considering avitaminosis A it is well to remember that food sources of vitamin A are limited. Aside from liver and a few oily fishes, man's sole dietary supply comes from the vegetable kingdom in the form of provitamin A or carotene which must be converted by the liver to the true vitamin before it can carry on the functions of vitamin A in the human economy.

This deficiency then may be due not

in 116 dermatologic patients and compared them with 116 control patients. The results were virtually identical. In six cases of Darier's disease two of pityriasis rubra pilaris and three of ichthy-

in vitamin A deficiency resembles acne vulgaris; however the lesions of acne vulgaris are pustular while in vitamin A deficiency this type of lesion is a rarity. The skin in acne is usually oily

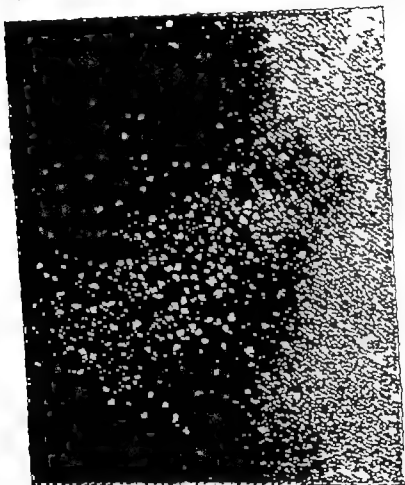


Fig. 663 Pityrioderma. Group of conical papules with central horny spines in twelve-year-old girl. (Courtesy of Dr. F. I. Faal.)

osis, the mean level of vitamin A was below normal. However in only two of the patients with Darier's disease did the skin become normal after treatment with vitamin A. The two with pityriasis rubra pilaris showed some but incomplete improvement.

Diagnosis. The papular eruption seen

while in vitamin A deficiency the skin is always dry.

Prophylaxis. Avitaminosis A is prevented by including the following articles in the diet: green leafy vegetables (spinach), yellow vegetables (carrots), eggs, milk, butter, apricots, yellow peaches, oranges, bananas, cod liver oil,

glands, and sebaceous glands of the skin. In short, these changes occur in all epithelial tissue and its potentiality as the first line of defense against invading organisms disappears.

Night Blindness (Nyctalopia) This is often the first subjective evidence of vitamin A deficiency.

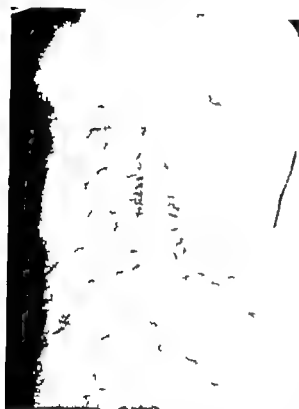


Fig. 661. Avitaminosis A. Night blindness is associated with the dermal manifestations.

Xerophthalmia. The specific effect of vitamin A deficiency is known as xerophthalmia. Keratinization of the epithelial lining of the tear glands and atrophy of these glands produce a red, denuded and edematous cornea which may lead to ulceration.

Cutaneous Lesions. The characteristic dermal manifestation of vitamin A deficiency is a dry, scaly skin with a papular eruption (phrynoderma). The hair follicles undergo hyperkeratosis and have the appearance of "goose flesh."

Vitamin A deficiency in infants and children also produces a dry, scaly skin, however without keratotic papules.

The characteristic nail changes consist of longitudinal ridges, transverse bands, and pitting. Vernix caseosa in infants often indicates vitamin A deficiency in the mother's diet.

Superficial erosion of the mucous membrane of the tongue and lower lip has been reported, and in the later stages of this disease a red, glazed tongue and excoriations of the angles of the mouth occur.

Since vitamin A plays an essential role in maintaining the integrity of all epithelial tissue, a deficiency is believed to increase the incidence of infection. The life span of the test animal on a diet totally deficient in vitamin A is materially reduced. At post mortem examination the most common condition in these animals is a lack of adipose tissue and a general visceral atrophy. Evidence of acute inflammation and infection is present at more than one site, the most common being abscesses at the base of the tongue. Other sites include kidney and bladder infection, acute inflammation of the intestines, sinuses, and middle-ear infections, and infection of the lungs. These findings are somewhat analogous to the symptoms of the more severe states of avitaminosis A met in clinical medicine. The dry, scaly skin, muscular weakness and wasting, diarrhea, corneal infection, ulceration, hypopyon with necrosis, superficial erosion of the mucous membrane of the tongue and lower lip, have all been reported as symptoms of avitaminosis A.

Infants with vitamin A deficiency are more prone to diaper dermatitis, intertrigo, moniliasis, and nutritional eczema.

Specific Diseases. Leitner and Moore determined the plasma, vitamin A levels

dose is advisable in the beginning; this treatment should be continued for several weeks and then gradually reduced. Treatment should continue from several months to a year.



Fig. 664 Phrynodermia. Generalized distribution of hyperkeratotic lesions in thirteen-year-old boy (Courtesy of Dr. Paul Faal.)

Avitaminosis D

Vitamin D is the term applied to any one of several related sterols which have antirachitic properties. It is present in the liver oils of various fish and is produced artificially by the activation of ergosterol and a few related sterols by their exposure to ultraviolet rays.

Avitaminosis D tends to cause rickets

in children and osteomalacia in adults. It is produced in the body on exposure to sunlight the chief dietary sources are cream, butter, liver and egg yolk. Cod liver oil, tuna fish liver oil, and certain other fish liver oils, and vitamin D milk are the best commercial sources for medicinal use.

Direct evidence that a deficiency of this vitamin causes any skin disease is lacking; however a deficiency may unfavorably influence allergic dermatoses, proriasis, pemphigus, neurodermatitis, acne vulgaris, and dental caries. A deficiency of vitamin D has been regarded by some as favoring the production of pemphigus and massive doses of 1,000,000 international units given daily have effected cures in some cases.

The Committee on Nutrition of the National Research Council has established the daily requirement for infants, children, and adolescents as being 600 international units. It is probably well to insure that this amount be consumed at all ages.

Avitaminosis B

Any discussion on the vitamins of the B complex at the present time should first of all consider them as an entity; secondly it should isolate the specific chemical substances of the group which have been shown to exert specific effect in clinical medicine; thirdly it should discuss those substances that have been isolated and identified but the clinical application for which has not been definitely established but which are believed to have some bearing on human nutrition.

Several of the factors of the B complex have been shown to be active fractions of enzymes which are essential for the breakdown of foodstuff in intracellular metabolism. Thiamine, nicotinic acid,

halibut liver oil and other fish liver oils, and liver. The daily optimum vitamin A requirement for an average adult is about 5000 international units. Increased

is materially reduced. It is well to fortify the diets of all such patients with some form of natural vitamin A. The one carrying the least animal fat should be

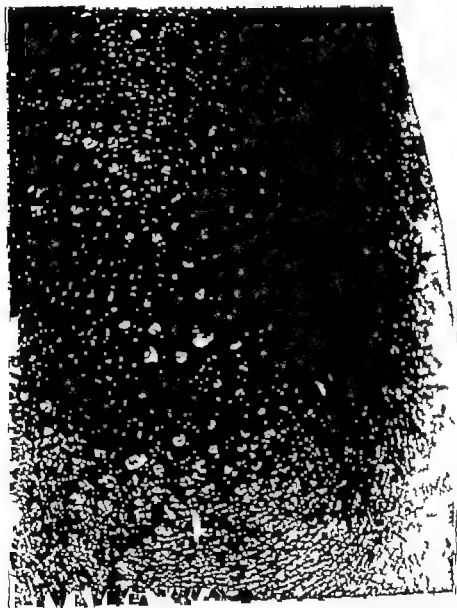


Fig. 665: Phrynoderma. Large flat papules, some with a central horny spine; skin of elbow in a fourteen-year-old boy (Courtesy of Dr. Paul Fama.)

amounts are recommended for pregnant and nursing mothers and during adolescence.

In patients with biliary disturbances and liver disease the utilization of the provitamin A of the vegetable kingdom

the one of choice a concentrate of fish liver oils is preferable.

Treatment Administration of a normal diet with the addition of 50 000 to 200 000 international units of vitamin A per day usually effects a cure. The larger

dose is advisable in the beginning; this treatment should be continued for several weeks and then gradually reduced. Treatment should continue from several months to a year.



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Several of the factors of the B complex have been shown to be active fractions of enzymes which are essential for the breakdown of foodstuff in intracellular metabolism. Thiamine, nicotinic acid,

and riboflavin have been shown to be essential constituents in many of the enzymes and coenzymes essential to the oxidative reduction mechanism by which the gradual release of energy of food is continuously and gradually liberated for use of the individual cell.

Rarely in clinical medicine do we find characteristic isolated deficiency of any single component of the B vitamins; commonly we find a more general state of nutritional failure superimposed upon which is the gross manifestation of one or more full blown specific factor deficiencies.

The classical example of this is pellagra for which nicotinic acid is most effective. The outstanding work of Spies has shown that the associated cheilitis which was not affected by nicotinic acid responded quite dramatically to the co-administration of riboflavin; that the associated polyneuritis and such symptoms as nervousness and insomnia responded only with the co-administration of thiamine and pyridoxin.

Dietary deficiency due to such factors as the poor handling of food for mass distribution, poor selection of available food, poor cooking such as the use of excessive water, overcooking or the prolonged constant application of heat such as is met wherever large groups are fed, make vitamin B deficiency the most common in clinical medicine.

Vitamin B compounds being water soluble are excreted in sweat and urine; therefore, any increase in total fluid intake or fluid loss, if continuous for any period of time should be accompanied with an increased administration of vitamin B. Examples of these conditions are the polyuria of diabetes, the administration of intravenous fluids, the forcing of fluids in urinary tract infections, and exposure to excessive tem-

peratures with resultant excessive perspiration (heat therapy). Laborers and men working under extremes of temperature should have their diet fortified by the daily administration of B complex.

Hyperthyroidism, fever, radiation therapy, pregnancy and lactation, and prolonged physical exertion or the delirium of certain psychoses require vitamin B complex.

The B-complex constituents that have been definitely shown as necessary in human nutrition are thiamine, riboflavin, nicotinic acid, folic acid and perhaps pyridoxine and pantothenic acid.

Thiamine Deficiency

The specific lesions resulting from thiamine deprivation (avitaminosis B₁) consist of degenerative changes in the myelin sheaths and nerve fibers.

The early manifestations of vitamin B₁ deficiency affecting peripheral nerves are pain and burning along the involved sensory neurons and impairment of motor nerve function. This involvement of the motor nerve function can occur in any part of the body and is the etiological factor in peripheral neuritis, loss of tone of the gastrointestinal tract, and the cardiac derangements occurring in beriberi. Loss of strength especially of quadriceps, tenderness of the calves, and hyperesthesia of the feet occur.

While specific dermatologic manifestations of thiamine deficiency do not occur it is well to remember that a deficiency of this substance may so interfere with the utilization of food that other deficiency states may occur.

Dosage. The Committee on Food and Nutrition of the National Research Council places the daily allowance for a moderately active man at 1.8 mg. Therapeutic dosage ranges from two to ten times this amount orally or parenterally.

Riboflavin Deficiency

Riboflavin is a heat-stable fraction of the B complex.

The most prominent lesion of riboflavin deficiency is cheilitis, or cheilosis, characterised by reddening of the lips with exfoliation of the epithelium and radiating fissures at the angles of the mouth. In chronic deficiency there is excessive and irregular wrinkling.

Symptoms The symptoms of riboflavin deficiency are

1. Macerated fissures, usually bilateral, occurring at the angles of the mouth.

2. Exaggerated redness, occurring on the lips along the line of closure. The lips, especially the lower frequently show an increase in vertical fissuring with exfoliation frequently causing a break in the mucous membrane.

3. A seborrheic syndrome characterized by a fine scaly slightly greasy desquamation on a slightly erythematous base occurring in the nasolabial folds, the alae nasi, the vestibule of the nose, and on the ears. The term *dysebacæa* has been used for this state.

4. The sebaceous glands are often the source of filiform excrescences varying in length up to 1 mm. These occur in greater or lesser numbers over the skin of the face although more frequently on the nasolabial folds and alae nasi.

5. Oral symptom consist of glossitis; the tongue is smooth the papillae flat or mushroom shaped, the color is purplish red or magenta hued rather than scarlet red as seen in nicotinic acid deficiency.

6. Eye symptoms are characterized by itching burning, a sensation of roughness on the conjunctivæ and mild photophobia. More rarely severe photophobia, dimness of vision and partial blindness may be present.

Pathology The pathology includes circumcorneal injection, congestion of the bulbar conjunctivæ, corneal vascularities, and corneal opacities. Skin and oral symptoms of ariboflavinosis usually disappear promptly while the ocular symptoms may require the administration of riboflavin over a period of months or even years. Riboflavin is found in yeast, milk, liver, wheat germ, eggs, cheese, leafy green vegetables, peas, lima beans, malt and muscle.

Dosage The daily requirement ranges from 2 to 3 mg. During pregnancy 4 to 6 mg is recommended. It is well to administer some form of the whole B complex where riboflavin deficiency predominates. The mouth lesions seen in nicotinic acid deficiencies are often associated with riboflavin deficiency. This combination of symptoms occurs in pellagra. In chronic ariboflavinosis, 5 to 6 mg three times daily is indicated for six months.

Nicotinic Acid Deficiency

Specific nicotinic acid (niacin) deficiency produces blacktongue in dogs and pellagra in man.

The skin of the pellagrin is characterized by a bilateral symmetrical dermatitis which may involve any part of the body but is more apt to occur on those areas commonly exposed to sunlight and trauma. The areas of predilection in the order of frequency are the dorsum of the hands, the wrists, the elbows, the dorsum of the feet, the ankles, the neck, the face, the submammary region, the inguinal and gluteal folds, the axillæ, and the perineum.

The early clinical changes consist of burning, tenderness, erythema, pigmentation and mild vesiculation on a slightly edematous base. These are followed, if untreated, by a regression of the erythema, the occurrence of desquamation and

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peratures with resultant excessive perspiration (heat therapy). Laborers and men working under extremes of temperature should have their diet fortified by the daily administration of B complex.

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While specific dermatologic manifestations of thiamine deficiency do not occur, it is well to remember that a deficiency of this substance may so interfere with the utilization of food that other deficiency states may occur.

Dosage. The Committee on Food and Nutrition of the National Research Council places the daily allowance for a moderately active man at 1.8 mg. Therapeutic dosage ranges from two to ten times this amount, orally or parenterally.

deficient diet become completely hairless, followed by a severe dermatitis. In rats, the loss of hair is most marked around the eyes and results in the so-called "spectacled eye" syndrome. Inositol occurs in all plant and animal tissue. The greatest amount occurs in the leaves of plants, citrus fruit, and cereals.

Pyridoxine Deficiency

Vitamin B₆ (pyridoxine) occurs largely as yeast, rice polishings, wheat, and fish. In fact, it is widely distributed over the entire animal and vegetable kingdom. Its absence causes acrodynia in rats, which is accompanied by edema and exhalosis. The effect of its absence in humans is not well known. Its administration to pellagrics hastens recovery. Chills may be a symptom of vitamin B₆ deficiency.

Avitaminosis C

Vitamin C is a water-soluble vitamin occurring in citrus fruits, strawberries, and other fruits, and in tomatoes, green peppers, paprika, potatoes, and other raw vegetables.

Vitamin C is readily oxidized. It is said that citrus fruit juice allowed to stand at room temperature for a single hour will lose an appreciable amount. Cooking frequently destroys the major part of the vitamin C of a well-selected meal. It has been said that the simple act of mashing and fluffing of potatoes destroys all of their vitamin C content. It is the perfect example of the difficult task of balancing a diet with food alone; the vitamin content of a well-selected diet can easily become a deficiency regimen through preparation alone.

Like the water-soluble B factors, vitamin C is washed from the body in any of those conditions involving excessive fluid intake or excessive fluid loss. The

classical example of vitamin C deficiency is scurvy. The cutaneous lesions of the disease in adults consist of follicular hyperkeratotic papules with or without perifollicular hemorrhage. In the more severe cases, every follicle may be involved. As a result of an increased capillary fragility there is often perifollicular hemorrhage, petechiae, and purpura. The skin in a scorbatic when traumatized will be the site of ecchymosis out of all proportion to the trauma causing it. The sites of predilection are the anterior and inner aspects of the thighs and the upper part of the legs. Lesions of the mouth are characteristic symptoms of scurvy. The gums are red, tender, swollen, boggy and bleed easily. The gums recede from the teeth and the interdental spaces are larger than normal. In chronic deficiency the receded gums are firm and thickened. The teeth undergo resorption and porosity of the dentine, while defects in the enamel and cementum have also been reported (Fish and Harris). Loosening and shedding of the teeth may eventually occur.

The constitutional symptoms of scurvy are weakness, palpitation, dyspnea, intestinal hemorrhages, edema of the joints (hydroarthrosis) and occasionally hematuria and fever.

Scurvy in infants usually occurs between the sixth and tenth months of life. It differs in no way from the scurvy of adults; however it is much more prevalent in adults. Scurvy does not occur in breast-fed infants and it has become infrequent since the addition of orange juice to bottle fed babies. The scorbatic infant is irritable, does not gain in weight, and the lower limbs are motionless and usually fixed on the abdomen. The limbs are tender and the slightest touch causes crying.

Diagnosis: The cutaneous lesions of

a more intense cutaneous pigmentation. The dermatitis is sharply demarcated from the adjacent normal skin occasionally an oozing of serum under the areas of desquamation is present. Lesions developing in other parts of the body are pathologically identical with the skin lesions.

The glossitis of pellagra is one of the most characteristic symptoms of this deficiency disease. The tongue is swollen, beefy red, smooth and dry in appearance. The papillae of the tongue, at first congested and hypertrophic, are later atrophic and associated with multiple fissuring. Secondary infection with Vincent's angina and monilia frequently occurs. The inflammatory process spreads to the buccal mucosa, the gums, lips, and pharynx. Deeply penetrating ulcers, especially painful, occur in the later stages.

The mucous membrane of the vagina and the rectum may be similarly affected. All of these symptoms, the dermal, the mucosal, as well as the characteristic diarrhea and dementia, respond promptly to nicotinic acid therapy. Improvement may be noted after a few days of treatment.

Dosage: Nicotinic acid amide is preferred to nicotinic acid because the latter frequently produces a vasodilatation that leads to a nondangerous but annoying flushing.

The Committee on Food and Nutrition of the National Research Council places the daily requirements for a moderately active man at 18 mg. For active treatment of pellagra, the dose is from 100 to 500 mg. daily. It should always be accompanied by a correction of the diet with a heavy protein intake. It is well to administer a reliable source of the whole B complex when nicotinic acid is used.

Biotin Deficiency

Experimental biotin deficiency in man on a diet in which about 30 per cent of the total calories was supplied by denatured egg white resulted in the occurrence of a number of clinical symptoms similar to those of spontaneous avitaminosis. A striking sallow pallor of the skin and mucous membranes appeared, followed by an increasing dryness of the skin with marked reticulation and a fine branny desquamation. Extreme lassitude and somnolence, muscle pains, precordial distress, and anorexia were observed. Parenteral administration of biotin cured these symptoms rapidly.

In man vitamin H therapy has given curative results in a few isolated cases of acne vulgaris, rosacea, and furunculosis. Certain cases of baldness in men are caused by seborrheic conditions and can be improved by vitamin H administration. More severe cases of seborrhea seem to be related to the skin disease called psoriasis, which consists of an eruption of circumscribed rounded patches, occurring chiefly on the elbows, knees, scalp, and back. Encouraging results have been attained in these cases by administration of vitamin H.

Pantothenic Acid Deficiency

Pantothenic acid has been identified as a factor in chick dermatitis and is an achromic factor in rats. In animal experimentation it has been shown that the absence of pantothenic acid produces alopecia and depigmentation of the fur. Its absence produces early senescence. The best sources are liver, kidney, rice, and bran. It is also produced by various molds and micro-organisms.

Inositol Deficiency

Inositol has also been shown to be antialopeic in mice. Mice on an inositol

deficient diet become completely hairless, followed by a severe dermatitis. In rats, the loss of hair is most marked around the eyes and results in the so-called spectacled eye syndrome. Inositol occurs in all plant and animal tissue. The greatest amount occurs in the leaves of plants, citrus fruit, and cereals.

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Diagnosis The cutaneous lesions of

avitaminosis C must be differentiated from those of avitaminosis A. The follicular lesions of avitaminosis A do not show follicular hemorrhages and in avitaminosis A there is usually xerophthalmia. The pathognomonic lesions of hypovitaminosis C are spongy and bleeding gums.

Prophylaxis. The best prophylactic measure is daily addition to the diet of fresh orange or lemon juice. In the absence of citrus fruit adults should be given 100 mg. of ascorbic acid daily and infants should be given 50 to 100 mg. daily. Prophylaxis in infants consists of the addition of one or two teaspoonfuls of orange juice to the daily diet.

Treatment. The treatment in advanced cases of scurvy consists in rest in bed and an abundant supply of vitamin C. In addition synthetic vitamin C must also be used.

Treatment for the spongy and bleeding gums consists of the frequent use of the following mouthwash:

Pul. alum.	13.0
Lemon juice	50.0
Mel rose et Soli horat N F q.s ad	500.0

Mild cases of hypovitaminosis C occurring in infants are cured by the administration of a teaspoonful of fresh orange juice every three or four hours for several days with the milk feedings.

Potato, which possesses rich antiscorbutic properties, is also given. The potato should be baked in its skin and then rubbed through a fine sieve and added to the milk in the proportion of two teaspoonfuls to 45 cc. (1½ ounces) of milk.

Avitaminosis E

Vitamin E is the fertility vitamin in nature, it occurs in the seed of certain plants. The most valuable natural source is wheat germ oil. It has been artificially produced as alphatocopherol.

No cutaneous symptoms have thus far been reported as due to a deficiency of this vitamin.

Avitaminosis K

Vitamin K is necessary for the normal absorption of prothrombin in the blood. Bile is necessary for its concentration and utilization. Its clinical application is to restore normal clotting time and prevent hemorrhage in operative procedures with bile obstruction (obstructive jaundice) and it is also administered prenatally to reduce hemorrhage in new born infants.

It is found in alfalfa, spinach, cabbage, purified fish meat, hog liver fat, egg yolk, and hemp seed.

For commercial use, it is extracted from alfalfa essentially. It has been produced artificially and introduced under the name "menadiolone."

No cutaneous symptoms, aside from a tendency to bleed, especially from minor wounds, have thus far been reported as due to a deficiency of this vitamin.

Avitaminosis P

Absence of vitamin P may produce petechiae and other hemorrhagic tendencies. It has a definite influence on capillary fragility. It is present in the skin of lemons, oranges, and grapefruit.

VITILIGO

SYNONYMS *Acquired leukoderma, achromia, piebald skin, leukopathia, leukismus.*



Fig. 665 Vitiligo.

Vitiligo is an affection of the skin characterized by white patches of skin and hair.

Incidence It rarely occurs before the tenth year or after the thirtieth. It occurs equally among males and females. It is found in tropical countries and in the dark-skinned races.

Etiology The exact cause is unknown. Vitiligo is sometimes hereditary and the majority of patients manifest neurocirculatory instability. It occurs in association with syphilis, myxedema, morphia, migraine, alopecia areata, Addison's disease, psoriasis, latent tuberculosis, lupus erythematosus, and chronic lead poisoning.

Temporary depigmentation of the hands and forearms has been observed in Negro workers who wore a certain brand of rubber gloves. The chemical respon-



Fig. 646 Vitiligo.

avitaminosis C must be differentiated from those of *avitaminosis A*. The follicular lesions of *avitaminosis A* do not show follicular hemorrhages and in *avitaminosis A* there is usually xerophthalmia. The pathognomonic lesions of *hypovitaminosis C* are spongy and bleeding gums.

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Pulv. lu	130
Lemon juice	600
Mel rose distilled borat N.F. q.s. ad	5000

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gold given once a week for a period of twelve weeks, has been favorably reported. "Covermark" or walnut juice (the fluid extract of fresh green hulls of black walnuts should be used) is the only method of disguising this condition.

The following is helpful

Fluident. about (Juglans)	10
Sol. curacine (F~)	20
in	
Arom. op. ammoniac	80
Alcohol q. ad.	1000
M. Apply several coats.	

The patches may also be tinted by using the following

I	
Tr. iodine	4.0
Spts. val. rect. q. s. ad.	80.0
Sca. dilute according to cosmetic indications, and apply	
II	
Titanium oxide	43.0
Calamine preparat.	43.0
Glycerine	10.0
Aq. rose q. s. ad	500.0
Sca. Add libthly of 10 to 20 drops, according to cosmetic indications, and apply	

sible was an antioxidant which on patch testing first produced an inflammation followed by depigmentation. The substance is known as *agerite alba* and is said to be a monobenzyl ether of hydroquinone. Gettel reported that hydroqui-



Fig. 667: Extensive Vitiligo. Syphiloderm. Vitiligo is generally limited to the neck, and is known as "the collar of Venus."

none used experimentally can turn hair gray (cat).

Pathology In the affected patches, the epidermis is devoid of pigment, otherwise, it is normal. In the surrounding areas the epidermis is hyperpigmented. The blood picture in vitiligo is not unlike that seen in lupus erythematosus.

Symptoms Vitiligo is characterized by patches of nonpigmented skin. The patches are round, oval, ivory white in tint, smooth, soft, and sharply defined. Increased pigmentation is usually present at the margin of the lesion. The sites of predilection are the dorsum of the hands, trunk, and neck. The patches enlarge slowly, often coalesce, and involve large areas. Hair in the affected

areas may or may not be depigmented. The course is progressive, however, it does not impair the general health. Exposure to sun or ultraviolet light increases the marginal pigmentation and makes the vitiligo more pronounced.

A depigmented areola may develop around a pigmented naevus and been termed by Sutton "leukoderma centrifugum acquisitum."

Diagnosis Vitiligo usually presents little difficulty in diagnosis. Albism, which may be partial or total, is a congenital anomaly and shows when total a lack of pigment in the eyes. The iris is pink and the choroid contains no pigment. In diagnosing vitiligo the personal history and serologic studies are necessary and may help.

Leukoderma colli due to syphilis (Fig. 667) is observed three to six or more months after a patient contracts syphilis. Circinate or oval depigmented spots appear on the neck, usually of about the same size and in variable number. The depigmented spots become most visible by looking at the neck tangentially. The diagnosis is completed by finding other signs and symptoms of syphilis.

Pseudovitiligo consists of two conditions: *achromia parasitica* and *non parasitic achromia*. In the first due to *trichomycosis*, microscopic examination reveals the fungus; in the second, the skin tans leaving islands not so pigmented or irregular macular depigmentation occurs as the tanning fades in the fall.

Treatment Treatment is not satisfactory. Counterirritation applied to areas of leukoderma may induce a certain amount of pigmentation. Strong doses of ultraviolet radiation may be used. The part previously painted with a 10 per cent alcoholic solution of the oil of bergamot. Painting the area with dilute Fowler's solution (1:10) is of benefit in some cases. The intravenous injection of

some other flat forms. The lipoidal deposit leads to hyperplasia of the connective tissue especially in nodular forms, with final almost complete change to a fibroma. In very old xanthoma, the growth is hard and, histologically such a lesion shows the structure largely of a

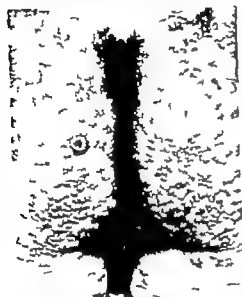


Fig. 670 Xanthoma Disseminatum. Colored male with blood cholesterol of 1780 mg. per 100 cc of blood. Xanthomatous papules universally distributed.

without hepatic disease hypercholesterolemia is present. Bloch and Schaeff attributed the lesions to an unbalance between the various lipoids. Cholesterol, on the basis of chemical analysis of the lesion, is a constant finding as are neutral fats (Weidman and Freeman). The lesions, therefore, are not merely cholesterol tumors nor are they necessarily connected with a high, blood-cholesterol content. It is, however possible that hypercholesterolemia is present at some stage in their evolution such variations in the blood lipids may account for the exacerbations or remission of the lesions in diabetic xanthoma. Weidman removed sections of tissue in patients with hypercholesterolemia (associated with nephritis, diabetes, and arteriosclerosis) but no xanthomas developed at the site of the injured tissues. It would, therefore appear that some unknown factor or factors, is necessary before mere lipid cutaneous deposits lead to the development of xanthomata.

Varieties Three forms of xanthoma are recognized namely xanthoma tuberosum multiplex, xanthoma palpebrarum, and xanthoma diabeticorum.

Xanthoma Tuberosum Multiplex. This form reveals widely disseminated papules, tubercles, or nodules (xanthoma disseminata and xanthoma tuberosum or en tumeurs). In xanthoma tuberosum, the sites of predilection are the elbows, hips, and knees. Streaked and ribbon-shaped lesions may appear on palms and soles. The term juvenile xanthoma is used for a form of xanthoma tuberosum multiplex seen in children. It is often familial, and many of the patients have cardiac disease. In xanthoma disseminata, the lemon-colored papules tend to become dark brown. They are localized to the extensor surfaces, axillae, popliteal and cubital areas, and neck. Diabetes

fibroma with characteristic xanthoma cells present only here and there. Diabetic xanthomas often disappear under suitable therapy before fibrous hyperplastic changes can occur.

Hypercholesterolemia occurs in patients with xanthoma, but it is not a constant finding nor is mere presence sufficient to explain the development of the lesions. According to Montgomery the blood lipids are within normal limits in disseminated xanthomatosis, with multiple small lesions predominating on the flexural surfaces and the mucosae but in ordinary xanthoma tuberosum, with r

XANTHOMA

SYNONYM: *Xanthelasma*.

Xanthoma is characterized by flat plaques, appearing as slightly elevated patches of yellow color (chamois-colored). The lesions are painless and usually lasting

tain syndrome Niemann Pick disease) occur more frequently without than with cutaneous manifestations. The skin lesions presumably follow the intracutaneous and subcutaneous deposition of cholesterol and related lipoids with resultant passive and productive reactions in and around the infiltrated cells or reticulo-histiocytic apparatus (change in color swelling development of lipid containing xanthoma or foam cells, multinucleated Touton type giant and foreign-



Fig. 668: Xanthoma Tuberosum

Etiology Xanthoma is now believed to be the localized or generalized cutaneous expression of a disturbance involving lipid metabolism. The role of at least some forms of hepatic disease of pituitary disease diabetes mellitus, and of diet in the development of the cutaneous lesions appears established. Biochemical and pathological studies however appear to indicate that lipid disturbances (Gaucher's disease Hand-Schüller Chris-



Fig. 669: Xanthoma Multiplex

body giant cells histologically and phenomena incident to the presence of a foreign body)

Aside from the lipid deposit, fibromatous changes are the rule in (nodular) xanthoma and less so in palpebral and

some other flat forms. The lipoidal deposit leads to hyperplasia of the connective tissue especially in nodular forms, with final, almost complete change to a fibroma. In very old xanthoma, the growth is hard and, histologically such a lesion shows the structure largely of a

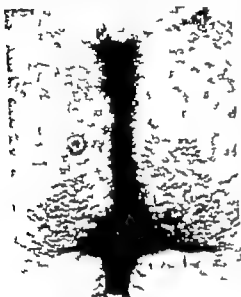


Fig. 679 Xanthoma Disseminatum. Colored scale with blood cholesterol of 1700 mg. per 100 cc. of blood. Xanthomatous papules universally distributed.

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insipidus is often associated and it may be the cutaneous expression of the Hand Schüller Christian syndrome

Xanthoma Planum and Palpebrarum.

This form appears on the eyelids, and much less commonly in the mouth and other parts of the body as closely aggregated yellow flattened nodules of

enched by trauma. Palmar and plantar carotinoderma is often present

Ninety per cent of patients with xanthoma diabeticorum are males and 80 per cent are between the ages of twenty and forty five years. Lesions may disappear after persisting for a few months or years. Relapses are common



Fig. 671 *Left* Xanthelasma. Note two large yellow plaques, one at each inner angle of both upper eyelids, and on plaque at inner angle right lower lid. *Right* Xanthelasma and Nodules of Xanthomatosis. In the same patient.

soft velvety texture. Xanthoma planum is not infrequently associated with other forms of xanthoma.

Xanthoma Diabeticorum. This form of xanthoma is characterized by firm discrete pinhead to pea sized chamois-colored papules. They are usually few in number but may be very abundant. They develop gradually or appear rather quickly and often at least in the early stage have a red inflammatory appearance. The lesions are occasionally tender, painful, and pruritic. The customary sites of predilection are the buttocks, elbows, and knees. The location of lesions is influ-

Xanthoma diabeticorum is more common in stocky middle-aged, diabetic males. Cardiovascular studies are indicated in all forms of xanthoma because of the frequency with which this system is involved.

Treatment. Xanthoma tuberosum multiplex is removed surgically. Xanthoma palpebrarum is obliterated by applications of trichloroacetic acid, carbon dioxide and the cautery. They may also be electrodesiccated or excised. The preferable procedure is surgical excision under anesthesia. Following infiltration with the anesthetic the yellow patch be-

comes prominent and can easily be pulled out with forceps or dissected out. If normal tissue has not been removed, sutures are usually unnecessary. A diet containing a minimum amount of fat is advised in xanthoma tuberosum multi-

plex and xanthoma palpebrarum. Chauffard suggests the following fat free diet: skim milk, fruits, vegetables, bread, and grilled meats. Xanthoma diabeticorum is controlled by adequate diet and insulin therapy.

XERODERMA PIGMENTOSUM (KAPOSI)

SYNONYMS *Melanosis lenticularis progressiva* (Pick) *atrophia dermatis pigmentosa* (Crocker)

This is a rare, congenital, often familial, cutaneous dystrophy the lesions of which appear as a result of exposure to light.

to ultraviolet rays but also to a variable degree to the x rays. Hematoporphyrin is not present in the blood.

Pathology The cutaneous degeneration appears somewhat analogous to that seen in senile degeneration of the skin, chronic arsenical skin, and especially to skin which has been subjected to prolonged and repeated exposure to x rays.



Fig. 672 Xeroderma Pigmentosum (Kaposi's disease). Note the numerous atrophic white spots, the lentiginosae, and the multiple epikelcosmata (both basal-cell and prickle-cell types).



Fig. 673 Xeroderma Pigmentosum. Note the numerous lentiginosae and the complicating basal-cell cancer of the lower eyelid.

Etiology The lesions commonly appear during the first three years of life, rarely after puberty. A history of consanguinity in the parents is obtained in almost 25 per cent of the patients, and the disease occurs about equally in the two sexes. The skin is hypersensitive not only

The cutaneous dystrophy becomes evident in predisposed persons under the influence of light.

Symptoms: The disease first becomes

apparent during the hot months, with attacks of erythema similar to that seen in ordinary sunburn. The areas involved are the uncovered ones: face, neck, upper chest, hands and forearms. As the apparent sunburn clears, tan spots, differing in no way clinically from lentigo, remain. These tend to darken, enlarge and multiply to the point where they appear almost confluent, yet discrete. The skin progressively becomes drier, darker and roughened, and the epidermis shows fine furfuraceous scales. Superficial ulcerations, covered with crusts and followed by scars, now develop. A variable number of telangiectases appear. The skin as a whole seems to undergo atrophy: is thinned but not adherent to the underlying structures; loses its elasticity. Conjunctivitis, traction of the lower lid (ectropion), narrowing of the buccal orifice and thinning of the nasal tip usually develop. At this stage the skin has a characteristic appearance as a result of the erythematous, pigmented and sclerotropic spots. As the patient ages, small verrucous elevations, which often undergo epitheliomatous degeneration, appear here and there on the pigmented and cicatricial areas. These may merely reach a certain size and then drop off, either spontaneously or as a result of washing or softening applications. In other instances they may vegetate and then ulcerate or simply ulcerate without first vegetating. These epitheliomas (basal or squamous cell)

either slowly or rapidly spread on the surface or in depth. Metastasis to the lymph nodes and early death follow.

Diagnosis. The diagnosis of xeroderma pigmentosum in fully developed cases is easy. Its gradual development in a young person, the localization of the lesions, their symmetry and the absence of a history of exposure to x-rays differentiate it from chronic *radiodermatitis*. *Lentigo* resembles xeroderma pigmentosum in the early stages, but the additional phenomena—xeroderma, telangiectases, and cicatricial spots—are absent. It is possible, however, that *lentigo* developing after repeated attacks of sunburn may be an abortive instance of this disease. In cutaneous *arsenicism* the pigmentation is diffuse and not macular.

Prognosis. Patients with this disease generally die young: from twelve to fifteen years is the usual life span, and rarely from puberty to twenty-five years. One of our patients lived to the age of thirty-two.

Treatment. The avoidance of sunlight undoubtedly prolongs the lives of these patients. The skin should be constantly covered with creams containing 2 per cent salol, quinine, calamine or tannic acid. When keratoses develop, they should immediately be destroyed by cautery or electrocoagulation. Solid carbon dioxide may be used. Epitheliomas should be excised or electrocoagulated.

YAWS

SYNONYMS *T. pertenue*, *frambesia*, *plan*, *lulus*, *songu*.

Yaws is a contagious, systemic disease characterized by a frambesiform (raspberrylike) eruption.

Varieties Three clinical stages are described (1) a primary stage characterized by a lesion at the site of inoculation, (2) a secondary stage, in which

colored races. It is not congenital or hereditary.

Etiology It is caused by the *Treponema pertenue*. Infection occurs through skin lesions. Flies and gnats are believed to carry the disease. It is transmitted readily from person to person by contact.



Fig. 675. Left Yaws (crab yaws). Right Yaws (ulcerohypertrophic). (Courtesy of Dr. V. Pardo Castella.)

known of yaws are more or less widely distributed over the skin, and (3) a tertiary stage which is characterized by ulcers and gummatous nodules.

Incidence Yaws is primarily a tropical disease. In the Western Hemisphere, it occurs in the West Indies, Mexico, Brazil, British Guiana, and Venezuela. Europeans are less susceptible than natives and it is largely confined to the

Symptoms The initial lesion of yaws is characterized by a raspberrylike papule usually located extragenitally. The period of incubation varies from two to four weeks. The lesion may be single or multiple. A yellowish secretion usually exudes from the papules, which dries completely or the papules may become nodular.

From one to three months after the

appearance of the initial lesion which is referred to as the "mother yaw" a secondary eruption occurs. The sites of predilection are the face, limbs, and mucocutaneous orifices. Lesions consist of granulomatous papules which resemble the primary lesion. On removal of the amber yellow crusts covering these papules, a red or reddish yellow fungating surface remains. The *Treponema pertenue* is easily demonstrable in this lesion. Occasionally the eruption is papular and scaly as well as ulcerative. The palms and soles may become pitted. Mucous membranes are seldom affected but the tongue is occasionally involved which is referred to as frametic leukoplakia. Alopecia and iritis do not occur.

In children the condition usually lasts three to six months, while in adults it lasts from six to twelve months. Recurrences are not uncommon. The blood Wassermann reaction is positive although in old cases it may be negative.

Tertiary symptoms include osteitis, periostitis, arthritis, contractures of muscles, dactylitis, occasionally ulcerated gummata of the skin, subcutaneous gangosa, paranasal tumors, and juxta-articular nodules. Fissures and ulcers occurring on the soles of the feet, known as "crab yaws" are very annoying and disabling in adults.

Prophylaxis Where yaws is endemic, contact with flies and other insects should be avoided. The patient should be isolated in a screened room at least during the secondary period of the eruptive process.

Treatment The intravenous use of *arsphenamine* is specific. *Bismuth* is probably less so. The initial and secondary lesions of yaws heal rapidly following a single injection of *arsphenamine*; however six weekly injections should be given in order to prevent recurrence. The tertiary lesions of yaws respond more slowly to therapy and a course of *arsenic* should be followed by a similar course of *bismuth*. *Potassium iodide* in doses of 1 gm (16 grains) in water three times daily is effective in tertiary yaws where leg ulcers and bone lesions are present.

Intramuscular injections of *penicillin* (40 000 units, every three hours, day and night for five days) cause a rapid disappearance of the cutaneous manifestations of yaws. The effects of *penicillin* alone upon the serologic evidences of the infection are still *sub judice*.

The results obtained by Hill and his associates, who used *penicillin* in oil (100 000 units daily for ten days) suggest that *penicillin* followed by a more prolonged *arsenical treatment* is the preferable method.

YEN SHE ECTHYMA

"Yen She" identifies the ash or residue in an opium smoker's pipe. Some opium habitues inject this residue subcutaneously or intramuscularly. Yen She

is insoluble. Nodules mark the sites of injection. Secondary infection follows these injections. Ecthymatous lesions are common sequelae.



Fig. 675 Yen She Ecthyma. Ecthymatous lesions following the hypodermic injection of "yen she," the ash remaining in the pipe of an opium smoker.

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